
MEDICO-CHIRURGICAL SOCIETY
EDINBURGH

THE TRANSACTIONS

OF THE

MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH.

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OF THE

MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH.

VOL. X.—NEW SERIES.

SESSION 1890-91.

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1891.

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PREFACE.

The present Volume is the *Tenth* of the *New Series*, and contains a record of the work done during the past Session.

That work, as hitherto, embraces the communication of Original Papers; the exhibition of Patients, illustrating rare and interesting forms of disease; and the exhibition of Pathological and other specimens, so essential to the proper understanding of the morbid changes which take place in the human body.

During the past Session one Extra Meeting was held. The evening was devoted to Clinical Instruction and the Exhibition of Pathological Specimens — when many interesting cases and specimens were exhibited. It is hoped that such Meetings will materially increase the usefulness of the Society.

It is believed that the publication of the Transactions in this permanent form will prove a valuable contribution to medical literature, will encourage the Members to take a more active part in the work of the Society, and will tend in no small degree to increase the influence and usefulness of the Medico-Chirurgical Society of Edinburgh.

William Craig,
Editor.

October 1891.

Medico-Chirurgical Society of Edinburgh.

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	John Haddon, M.D., C.M., Marsh House, Canonbie . .	1883
	* German Sims Woodhead, M.D., F.R.C.P. Ed., Beverley, Nightingale Lane, Balham, London, . . .	1883
	Thomas Francis Spittal Caverhill, M.B., F.R.C.P. Ed., 8A Abercromby Place, ...	1883
	Robert Alexander Lundie, M.B., B.Sc., F.R.C.S. Ed., 55A Grange Road, ...	1883
140	Professor Arthur W. Hare, M.B., F.R.C.S. Ed., M.R.C.S. Eng., 23 St John Street, Manchester, ...	1883
	Edwin Baily, M.B., C.M., Oban, ...	1883
	Alexander Black, M.B., F.R.C.P. Ed., 13 Howe Street, .	1883

		Date of Admission.
	Harry Melville Dunlop, M.D., F.R.C.P. Ed., 20 Abercromby Place,	1883
	George Andreas Berry, M.B., F.R.C.S. Ed., 31 Drumsheugh Gardens,	1883
145	Hamilton Wylie, M.B., C.M., 1 George Place,	1883
	Arthur Douglas Webster, M.D., F.R.C.P. Ed., 20 Newington Road,	1883
	Robert William Philip, M.D., F.R.C.P. Ed., 4 Melville Crescent,	1883
	Joseph Carne Ross, M.D., F.R.C.P. Ed., 98 Mosley Street, Manchester,	1884
	William Russell, M.D., F.R.C.P. Ed., 46 Albany Street, *Secretary*,	1884
150	George Dickson, M.D., F.R.C.S. Ed., 9 India Street,	1884
	Thomas Wyld Pairman, L.R.C.P. & S. Ed., H. M. Prison, Lyttelton, New Zealand,	1884
	Alexander Thom, M.D., C.M., Crieff,	1884
	Hugh Logan Calder, M.D., C.M., 60 Leith Walk,	1884
	James Craig Balfour, L.R.C.P. & S. Ed., West Street, Belford, Northumberland,	1884
155	Frederick Anastasius Saunders, L.R.C.P. Ed., F.R.C.S. Ed., Denburn, Crail,	1884
	Wm. Richardson, M.D., F.R.C.S. Ed., Bath Lodge, Reading,	1884
	Andrew Brown, M.D., M.R.C.P. Ed., 1 Bartholomew Road, Kentish Town, London, N.W.	1884
	G. J. H. Bell, M.B., C.M., Surgeon, Bengal Army,	1884
	T. Goodall Nasmyth, M.D., D.Sc., Cowdenbeath, Fife,	1884
160	Henry Hay, M.B., C.M., 7 Brandon Street,	1884
	Thomas R. Scott, M.D., C.M., Musselburgh,	1884
	R. Milne Murray, M.B., F.R.C.P. Ed., 10 Hope Street,	1884
	A. S. Cumming, M.D., F.R.C.P. Ed., 18 Ainslie Place,	1884
	Ernest F. Neve, M.D., F.R.C.S. Ed., M.R.C.S. Eng., Hospital, Srinagar, Kashmir, N.W. India,	1884
165	W. C. Greig, M.B., C.M., Tangier, Morocco,	1884
	John Mowat, M.D., 1 Hope Park Terrace,	1885
	Skene Keith, M.B., F.R.C.S. Ed., 42 Charles Street, Berkeley Square, London, W.,	1885
	D. Noël Paton, M.D., F.R.C.P. Ed., 4 Walker Street,	1885
	George Hugh Mackay, M.B., C.M., Elgin,	1885
170	Michael Dewar, M.D., C.M., 24 Lauriston Place,	1885
	Edward M'Callum, F.R.C.S. Ed., 3 Brandon Street,	1885
	T. Edgar Underhill, M.D., F.R.C.S. Ed., Bromsgrove, Worcestershire,	1885
	John Struthers Stewart, L.R.C.P. & S. Ed., 16 Merchiston Terrace,	1885
	Allen Thomson Sloan, M.D., C.M., 22 Forth Street,	1885
175	John William Ballantyne, M.D., F.R.C.P. Ed., 24 Melville St.,	1885
	James Robertson Crease, F.R.C.S. Ed., 2 Ogle Terrace, South Shields,	1885
	George Kerr, M.B., C.M., 6 St Colme Street,	1885
	Tom Bairstow, L.R.C.P. & S. Ed., 14 Buccleuch Place	1885
	David Milligan, M.B., C.M., 11 Palmerston Place,	1885
180	George Dods, M.D., L.R.C.S. Ed., 50 Great King Street,	1885
	J. Murdoch Brown, M.B., F.R.C.P. Ed., 9 Walker Street,	1885
	Robert W. Felkin, M.D., F.R.C.S. Ed., 20 Alva Street,	1885
	S. Hale Puckle, M.B., C.M., Bishop's Castle, Shropshire,	1885
	James Haig Ferguson, M.D., F.R.C.P. Ed., M.R.C.S. Eng., 25 Rutland Street,	1885
185	Charles Kennedy, M.D., C.M., 25 Newington Road,	1886
	William Gayton, M.D., M.R.C.S. Eng., Bartram Lodge, Fleet Road, Hampstead, London, N.W.,	1886
	Reginald Ernest Horsley, M.B., F.R.C.S. Ed., 46 Heriot Row,	1886

		Date of Admission.
	James Mill, M.B., C.M., 178 Ferry Road,	1886
	Robert Fraser Calder Leith, M.B., B.Sc., M.R.C.P. Ed., 129 Warrender Park Road,	1886
190	Thomas M. Burn-Murdoch, M.B., C.M., 31 Morningside Road,	1886
	Professor William Smith Greenfield, M.D., F.R.C.P. Lond. and Ed., 7 Heriot Row,	1886
	Oswald Gillespie Wood, M.D., F.R.C.S. Ed., Surgeon, Army Medical Staff, India,	1886
	James Hogarth Pringle, M.B., C.M., 5 Livingstone Place.	1886
	Nathaniel Thomas Brewis, M.B., F.R.C.P. Ed., 23 Rutland St.,	1886
195	John Batty Tuke, jr., M.D., F.R.C.P. Ed., Balgreen, Murrayfield,	1886
	David Berry Hart, M.D., F.R.C.P. Ed., 29 Charlotte Square,	1886
	Walter Scott Lang, M.D., F.R.C.S. Ed., M.R.C.S. Eng., Edinburgh,	1886
	Alfred Bell Whitton, M.B., C.M., Aberchirder,	1886
	Robert S. Aitchison, M.B., F.R.C.P. Ed., 83 Great King Street,	1887
200	J. A. Armitage, M.D., C.M., 15 Waterloo Road, Wolverhampton,	1887
	J. Walton Hamp, L.F.P.S. Glasg., L.S.A. Lond., Wolverhampton,	1887
	William Hunter, M.D., M.R.C.S. Eng., M.R.C.P.L., 61 Wimpole Street, Cavendish Square, London, W.,	1887
	Sydney Rumboll, L.R.C.P. Ed., F.R.C.S. Ed., Belgaum House, Leeds,	1887
	John Thomson, M.B., F.R.C.P. Ed., 14 Coates Crescent,	1887
205	George Franklin Shiels, M.D., C.M., 229 Geary Street, San Francisco,	1887
	T. Brown Darling, M.D., C.M., 165 Bruntsfield Place,	1887
	John Keay, M.B., M.R.C.P. Ed., Mavisbank House, Polton,	1887
	John F. Sturrock, M.B., C.M., Homewood, Broughty Ferry,	1887
	Edward Carmichael, M.D., 12 London Street,	1887
210	Charles C. Teacher, M.B., C.M., 16 Newington Road,	1887
	David W. Aitken, M.B., C.M., 17 Hatton Place,	1887
	Robert Inch, M.B., C.M., Gorebridge,	1887
	John Shaw M'Laren, M.B., F.R.C.S. Ed., 14 Walker Street,	1887
	George Mackay, M.D., F.R.C.S. Ed., M.R.C.S. Eng., 2 Randolph Place,	1887
215	Henry Alexis Thomson, M.D., F.R.C.S. Ed., 2 Coates Crescent,	1887
	David Wallace, M.B., F.R.C.S. Ed., 66 Northumberland Street,	1887
	John C. Messer, M.D., R.N., 15 Belgrave Place,	1887
	D. H. Anderson, M.B., C.M., Ulverston, Lancashire,	1887
	James Lockhart Wilson, M.B., C.M., Duns,	1888
220	William Booth, F.R.C.S. Ed., 2 Minto Street,	1888
	John M'Fadyean, M.B., C.M., 9 East Hermitage Place, Leith,	1888
	Thomas Russell, L.F.P.S. Glasg., 27A Westmuir Street, Parkhead, Glasgow,	1888
	John Ross Home Ross, M.B., F.R.C.P. Ed., 40 York Place,	1888
	George M. Johnston, M.D., C.M., 9 Morton Street, Leith,	1888
225	George Pirrie Boddie, M.B., C.M., 147 Bruntsfield Place,	1888
	Kenneth Mackinnon Douglas, M.D., F.R.C.S. Ed., 32 Alva Street,	1888
	George Lovell Gulland, M.D., F.R.C.P. Ed., 6 Randolph Place,	1888
	William Burns Macdonald, M.B., C.M., Port Lodge, Dunbar,	1888
	James Williamson Martin, M.D., F.R.C.P. Ed., 59 Ferry Road, Leith,	1888
230	Charles H. Bedford, M.B., B.Sc., M.R.C.S. Eng., H. M. Bengal Army, 55 George Square,	1889
	William Haldane, M.D., F.F.P.S. Glasg., Viewforth, Bridge of Allan,	1889
	John Hugh Alex. Laing, M.B., C.M., 11 Melville Street,	1889
	Harold Jalland Stiles, M.B., F.R.C.S. Ed., 5 Castle Terrace,	1889

		Date of Admission.
	John Smith, M.D., M.R.C.S. Eng., Bryceball, Kirkcaldy,	1889
235	Allan Cuthbertson Sym, M.D., C.M., 144 Morningside Road,	1889
	Edmund Frederick Tanney Price, M.B., C.M., 28 Mayfield Road,	1889
	John Berry Haycraft, M.D., Sc.D., 20 Ann Street,	1889
	Henry Harvey Littlejohn, M.B., F.R.C.S. Ed., 13 Victoria Road, Sheffield,	1889
	Albert Edward Morison, M.B., F.R.C.S. Ed., M.R.C.S. Eng., Brougham Terrace, Hartlepool,	1889
240	William George Sym, M.D., F.R.C.S. Ed., 58 Queen Street,	1889
	Benjamin D. C. Bell, L.R.C.P. and S. Ed., Kirkwall,	1889
	Hugh Jamieson, M.B., C.M., care of A. W. Hutton, Esq., M.A., 479 Avenida Montesdeoca, Buenos Ayres,	1889
	David Gair Braidwood, M.B., C.M., Halkirk, Caithness,	1889
	A. Home Douglas, M.B., M.R.C.P. Ed., 6 W. Maitland St.,	1889
245	Alexander John Keiller, L.R.C.P. and S. Ed., 21 Queen Street,	1889
	G. Keppie Paterson, M.B., C.M., 17 Forth Street,	1889
	William Stewart, M.D., F.F.P.S. Glasg., 146 Ferry Road, Leith,	1889
	Alfred William Hughes, M.B., F.R.C.S. Ed., M.R.C.S. Eng., Woodside, Musselburgh,	1889
	Thomas Proudfoot, M.B., M.R.C.P. Ed., 13 Lauriston Place,	1889
250	William H. Barrett, M.B., C.M., 21 Learmonth Terrace,	1890
	Dawson Fyers Duckworth Turner, M.D., F.R.C.P. Ed., 7 George Square,	1890
	Edward Farr Armour, M.B., C.M., 149 Bruntsfield Place,	1890
	James Hunter, M.D., C.M., Linlithgow,	1890
	William Guy, L.R.C.P. and S. Ed., 11 Wemyss Place,	1890
255	William Smith, L.R.C.P. and S. Ed., L.F.P.S. Glasg., Almond Bank, Midcalder,	1890
	Robert A. Fleming, M.B., C.M., M.R.C.P. Ed., 36 Drumsheugh Gardens,	1890
	Robert Thin, M.B., C.M., 6 Albany Street,	1890
	George M. Robertson, M.B., M.R.C.P. Ed., Morningside Royal Asylum,	1890
	John Mackintosh Balfour, M.B., M.R.C.P. Ed.,	1890
260	James Hutcheson, M.D., F.R.C.S. Ed., 8 Nelson Street,	1890
	A. Cowan Guthrie, M.B., C.M., 53 Charlotte Street, Leith,	1890
	Ralph Stockman, M.D., F.R.C.P. Ed., 12 Hope Street,	1891
	Alexander Lockhart Gillespie, M.B., M.R.C.P. Ed., 10 Walker Street,	1891
	Stewart Stirling, M.D., F.R.C.S. Ed., 6 Clifton Terrace,	1891
265	Francis D. Boyd, M.B., C.M., 3 Coates Place,	1891

NON-RESIDENT.

	Arthur Edward Turnour, M.D., M.R.C.S. Eng., *Denbigh*,	1843
	W. Ord M'Kenzie, M.D., L.R.C.S. Ed., *London*,	1845
	W. Judson Van Someren, M.D., L.R.C.S. Ed., *Redhill, Surrey*,	1845
	William H. Lowe, M.D., F.R.C.P. Ed., *Lincoln*,	1845
270	George Skene Keith, M.D., F.R.C.P. Ed., *Currie*,	1845
	Veitch Sinclair, L.R.C.P. and S. Ed., *London*,	1850
	Archibald Hall, M.D., *Montreal*,	1853
	W. Overend Priestley, M.D., LL.D., F.R.C.P. Ed., *London*,	1854
	Horatio Robinson Storer, M.D., *Newport, Rhode Island, U.S.*,	1855
275	James C. Howden, M.D., *Montrose*,	1856
	Thomas Skinner, M.D., L.R.C.S. Ed., *London*,	1856
	Professor William Smoult Playfair, M.D., LL.D., F.R.C.P.L., *London*,	1857
	J. Ivor Murray, M.D., F.R.C.S. Ed., *Scarboro'*,	1857
	Andrew Scott Myrtle, M.D., L.R.C.S. Ed., *Harrogate*,	1859
280	Robert Foulis, M.D., F.R.C.S. Ed., *Cupar-Fife*,	1859
	Francis Robertson Macdonald, M.D., *Inveraray*,	1860

		Date of Admission.
	Professor John Young, M.D., *University of Glasgow*, . .	1860
	Norman Bethune, M.D., F.R.C.S. Ed., *Toronto*, . .	1861
	George Thin, M.D., L.R.C.S. Ed., *London*, . . .	1861
285	J. Cecil Phillippo, M.D., *Kingston, Jamaica*, . . .	1861
	Professor William Stephenson, M.D., F.R.C.S. Ed., *Aberdeen*,	1861
	J. S. Beveridge, M.R.C.P. Lond., F.R.C.S. Ed., *Edenbridge*, .	1861
	David Yellowlees, M.D., LL.D., F.F.P.S. Glasg., *Glasgow*, .	1862
	Prof. Arthur Gamgee, M.D., F.R.C.P. Ed., F.R.S., *London*, .	1864
290	Professor John Cleland, M.D., LL.D., *The University, Glasgow*,	1864
	R. B. Finlay, M.D., Q.C., M.P., *Middle Temple, London*, .	1864
	Stanley Lewis Haynes, M.D., M.R.C.S. Eng., *Malvern*, .	1864
	Francis D. A. Skae, M.D., *Lerwick*, ...	1864
	James Watt Black, M.D., F.R.C.P.L., *London*, . .	1865
295	David Brodie, M.D., ...	1865
	Peter Maury Deas, M.B., L.R.C.S. Ed., *Exeter*, . .	1868
	Professor J. G. M'Kendrick, M.D., LL.D., F.R.C.P. Ed., *University, Glasgow*, ...	1870
	Professor Lawson Tait, M.D., F.R.C.S. Ed. and Eng., LL.D., *Birmingham*, ...	1870
	J. G. Sinclair Coghill, M.D., F.R.C.P. Ed., *Ventnor*, . .	1870
300	James Johnston, M.D., F.R.C.S. Ed., *London*, . .	1871
	J. William Eastwood, M.D., M.R.C.P.L., *Darlington*, .	1871
	Professor J. Bell Pettigrew, M.D., LL.D., F.R.C.P. Ed., *University of St Andrews*, ...	1873
	J. Johnson Bailey, M.D., F.R.C.S. Ed., *Marple*, . .	1874
	John Aymers Macdougall, M.D., F.R.C.S. Ed., *France*, .	1875
305	Thomas John Maclagan, M.D., M.R.C.P.L., *London*, . .	1875
	Dr Groesbeck, *Cincinnati*, ...	1875
	Professor David James Hamilton, M.B., F.R.C.S. Ed., *Aberdeen University*, ...	1876
	J. Moolman, M.B., C.M., *Cape of Good Hope*, . . .	1877
	Robert Somerville, M.D., F.R.C.S. Ed., *Galashiels*, . .	1877
310	Graham Steell, M.D., F.R.C.P.L., *Manchester*, . .	1877
	Frederick William Barry, M.D., D.Sc., *London*, . .	1878
	John Brown, M.D., F.R.C.S. Eng., *Burnley*, . . .	1878
	Keith Norman Macdonald, M.D., F.R.C.P. Ed., *Skye*, .	1880
	John Home Hay, M.D., M.R.C.S. Eng., *Alloa*, . .	1880
315	John Mackay, M.D., L.R.C.S. Ed., *Aberfeldy*, . . .	1881

ORDINARY MEMBERS

ARRANGED ALPHABETICALLY.

RESIDENT.

	Dr J. O. Affleck, 38 Heriot Row, ...	1871
	Dr R. S. Aitchison, 83 Great King Street, . . .	1887
	Dr D. Aitken, 17 Hatton Place, ...	1887
	Dr D. H. Anderson, Ulverston, Lancashire, . . .	1887
5	Dr James Andrew, 2 Atholl Crescent, ...	1869
	Professor Annandale, 34 Charlotte Square, . . .	1863
	Dr Archibald, Woodhouse-Eaves, Loughborough, . .	1882
	Dr J. A. Armitage, 15 Waterloo Road, Wolverhampton, .	1887
	Dr E. F. Armour, 149 Bruntsfield Place, . . .	1890
10	Dr W. Badger, Penicuik, ...	1882
	Dr Edwin Baily, Oban, ...	1883
	Tom Bairstow, Esq., 14 Buccleuch Place, . . .	1885
	Dr Andrew Balfour, Portobello, ...	1874
	Dr J. H. Balfour, Portobello, ...	1881
15	Dr G. W. Balfour, 17 Walker Street, ...	1874
	Dr James Craig Balfour, West Street, Belford, Northumberland,	1884

		Date of Admission.
	Dr J. M. Balfour,	1890
	Dr Thomas Balfour, 51 George Square,	1856
	Dr Alexander Ballantyne, Dalkeith,	1872
20	Dr J. W. Ballantyne, 24 Melville Street,	1885
	Dr A. H. Freeland Barbour, 8 Melville Crescent,	1881
	Dr W. H. Barrett, 21 Learmonth Terrace,	1890
	Surgeon C. H. Bedford, M.B., H. M. Bengal Army, Indian Medical Service, 55 George Square,	1889
	Dr Benjamin D. C. Bell, Kirkwall,	1889
25	Dr G. J. H. Bell, Surgeon, Bengal Army,	1884
	Joseph Bell, Esq., 2 Melville Crescent,	1862
	G. H. Bentley, Esq., Kirkliston,	1877
	Dr G. A. Berry, 31 Drumsheugh Gardens,	1883
	Dr Alexander Black, 13 Howe Street,	1883
30	Dr W. T. Black, 2 George Square,	1877
	Dr Robert H. Blaikie, 42 Minto Street,	1883
	Dr Bleloch, 2 Lonsdale Terrace,	1871
	Dr G. P. Boddie, 147 Bruntsfield Place,	1888
	William Booth, Esq., 2 Minto Street,	1888
35	Dr F. D. Boyd, 3 Coates Place.	1891
	Dr D. G. Braidwood, Halkirk, Caithness,	1889
	Dr Brakenridge, 10 St Colme Street, *Vice-President*,	1865
	Dr Byrom Bramwell, 23 Drumsheugh Gardens,	1876
	Dr N. T. Brewis, 23 Rutland Street,	1886
40	Dr Brown, 1 Bartholomew Road, Kentish Town, London, N.W.,	1884
	Dr J. Graham Brown, 16 Ainslie Place,	1878
	Dr J. Macdonald Brown, 12 South Mansionhouse Road,	1883
	Dr J. Murdoch Brown, 9 Walker Street,	1885
	Dr Alexander Bruce, 13 Alva Street,	1883
45	Dr Buist, 1 Clifton Terrace,	1877
	Dr T. M. Burn-Murdoch, 31 Morningside Road,	1886
	Dr Cadell, 22 Ainslie Place,	1870
	Dr Francis M. Caird, 21 Rutland Street,	1883
	Dr H. L. Calder, 60 Leith Walk,	1884
50	Dr W. Watson Campbell, Duns,	1877
	Dr Cappie, 37 Lauriston Place,	1855
	Dr Edward Carmichael, 12 London Street,	1887
	Dr J. Carmichael, 22 Northumberland Street,	1870
	Dr C. W. Cathcart, 8 Randolph Crescent,	1883
55	Dr T. F. S. Caverhill, 8A Abercromby Place,	1884
	Professor John Chiene, 26 Charlotte Square,	1867
	Dr Church, 36 George Square,	1876
	Dr Clouston, Tipperlinn House, Morningside Place,	1861
	Dr A. R. Coldstream, Florence, Italy,	1878
60	Dr John Connel, Peebles,	1876
	Dr Cotterill, 24 Manor Place,	1878
	Dr William Craig, 71 Bruntsfield Place,	1869
	Dr J. R. Crease, 2 Ogle Terrace, South Shields,	1885
	Dr Halliday Croom, 25 Charlotte Square,	1870
65	Dr A. S. Cumming, 18 Ainslie Place,	1884
	Dr R. J. B. Cunynghame, 18 Rothesay Place,	1868
	Dr T. B. Darling, 165 Bruntsfield Place,	1887
	Dr M. Dewar, 24 Lauriston Place,	1885
	Dr Archibald Dickson, Hartree House, Biggar,	1871
70	Dr George Dickson, 9 India Street,	1884
	Dr George Dods, 50 Great King Street,	1885
	Dr Halliday Douglas, 30 Melville Street,	1842
	Dr A. Home Douglas, 6 West Maitland Street,	1889
	Dr Kenneth M. Douglas, 32 Alva Street,	1888
75	Dr William B. Dow, Dunfermline,	1879
	Dr John Duncan, 8 Ainslie Place,	1868
	Dr Kirk Duncanson, 22 Drumsheugh Gardens,	1871
	Dr H. M. Dunlop, 20 Abercromby Place,	1883

		Date of Admission.
	Dr J. Dunsmure, 53 Queen Street,	1872
80	C. H. Fasson, Esq., Dep. Surg.-Gen., Royal Infirmary,	1879
	Dr R. W. Felkin, 20 Alva Street,	1885
	Dr J. Haig Ferguson, 25 Rutland Street,	1885
	Dr W. A. Finlay, St Helen's, Russell Place, Trinity,	1875
	Dr Andrew Fleming, 8 Napier Road,	1880
85	Dr R. A. Fleming, 36 Drumsheugh Gardens,	1890
	Dr Foulis, 34 Heriot Row,	1875
	Dr F. W. Dyce Fraser, Gorton House, Lasswade,	1883
	Dr John Fraser, 19 Strathearn Road,	1878
	Professor Thomas R. Fraser, 13 Drumsheugh Gardens,	1865
90	Dr R. Freeland, Broxburn,	1879
	Dr Garland, 35 Charlotte Street, Leith,	1873
	Dr W. Gayton, Bartram Lodge, Fleet Road, Hampstead, London, N.W.,	1886
	Dr G. A. Gibson, 17 Alva Street,	1880
	Dr James D. Gillespie, 10 Walker Street,	1852
95	Dr A. Lockhart Gillespie, 10 Walker Street,	1891
	G. R. Gilruth, Esq., 48 Northumberland Street,	1869
	Dr J. Allan Gray, 107 Ferry Road,	1879
	Professor Greenfield, 7 Heriot Row,	1886
	Dr David Greig, 38 Coates Gardens,	1854
100	Dr W. C. Greig, Tangier, Morocco,	1884
	Dr G. L. Gulland, 6 Randolph Place,	1888
	His Excellency Dr R. H. Gunning, 12 Addison Crescent, West Kensington, London, W.,	1846
	Dr A. C. Guthrie, 53 Charlotte Street, Leith,	1890
	Dr William Guy, 11 Wemyss Place,	1890
105	Dr John Haddon, Marsh House, Canonbie,	1883
	Dr William Haldane, Viewforth, Bridge of Allan,	1889
	Dr J. W. Hamp, Wolverhampton,	1887
	Professor A. W. Hare, 23 St John Street, Manchester,	1883
	Dr D. Berry Hart, 29 Charlotte Square,	1886
110	Dr Henry Hay, 7 Brandon Street,	1884
	Dr J. Berry Haycraft, 20 Ann Street,	1889
	Dr John Henderson, 7 John's Place, Leith,	1848
	Dr J. W. B. Hodsdon, 30 Walker Street,	1883
	Dr R. E. Horsley, 46 Heriot Row,	1886
115	Dr A. W. Hughes, Woodside, Musselburgh,	1889
	Dr George Hunter, 33 Palmerston Place,	1876
	Dr James Hunter, St Catherine's, Linlithgow,	1890
	Dr W. Hunter, 61 Wimpole Street, Cavendish Square, London, W.,	1887
	Dr Husband, 4 Royal Circus,	1849
120	Dr J. Hutcheson, 8 Nelson Street,	1890
	Francis B. Imlach, Esq., 48 Queen Street,	1843
	Dr Robert Inch, Gorebridge,	1887
	Dr W. Wotherspoon Ireland, Prestonpans,	1883
	Dr James, 44 Melville Street,	1877
125	Dr Allan Jamieson, 35 Charlotte Square,	1876
	Dr Hugh Jamieson, care of A. W. Hutton, Esq., M.A., 479 Avenida Montesdeoca, Buenos Ayres,	1889
	Dr James Jamieson, 43 George Square,	1877
	Dr G. M. Johnston, 9 Morton Street, Leith,	1888
	Dr R. M'Kenzie Johnston, 44 Charlotte Square,	1883
130	Dr J. Carlyle Johnstone, Melrose Asylum, Melrose,	1882
	Dr J. Keay, Mavisbank House, Polton,	1887
	Dr Keiller, 21 Queen Street,	1845
	Dr A. J. Keiller, 21 Queen Street,	1889
	Dr Skene Keith, 42 Charles Street, Berkeley Square, London, W.,	1885
135	Dr Thomas Keith, 42 Charles Street, Berkeley Square, London, W.,	1852
	Dr C. Kennedy, 25 Newington Road,	1886

		Date of Admission.
	Dr George Kerr, 6 St Colme Street,	1885
	Dr J. H. A. Laing, 11 Melville Street,	1889
	Dr W. Scott Lang, Edinburgh,	1886
140	Dr Robert Lawson, 24 Mayfield Terrace,	1881
	Dr R. F. C. Leith, 129 Warrender Park Road,	1886
	Dr George Leslie, Falkirk,	1881
	Dr Linton, 60 George Square,	1863
	Dr Littlejohn, 24 Royal Circus,	1853
145	Dr Harvey Littlejohn, 13 Victoria Road. Sheffield,	1889
	Dr Lucas, Dalkeith,	1875
	Dr R. A. Lundie, 55A Grange Road,	1883
	Dr P. M'Bride, 16 Chester Street,	1879
	Dr E. M'Callum, 3 Brandon Street,	1885
150	Dr W. Burn Macdonald, Port Lodge, Dunbar,	1888
	Dr J. M'Fadyean, 9 East Hermitage Place, Leith.	1888
	John M'Gibbon, Esq., 55 Queen Street,	1868
	Dr MacGillivray, 11 Rutland Street,	1877
	Dr G. Mackay, 2A Gilmore Place,	1878
155	Dr George Mackay, 2 Randolph Place,	1887
	Dr G. H. Mackay, Elgin,	1885
	Professor Sir Douglas Maclagan, 28 Heriot Row,	1834
	Dr J. S. M'Laren, 14 Walker Street,	1887
	Dr P. H. Maclaren, 1 Drumsheugh Gardens, *Vice-President*,	1868
160	Dr Roderick M'Laren, 23 Portland Square, Carlisle,	1882
	Dr J. W. Martin, 59 Ferry Road, Leith,	1888
	Dr A. Matthew, Corstorphine,	1882
	Dr D. Menzies, 20 Rutland Square,	1878
	Dr J. C. Messer, 15 Belgrave Place,	1887
165	Dr J. Mill, 178 Ferry Road,	1886
	A. G. Miller, Esq., 7 Coates Crescent, *Vice-President*,	1867
	Dr D. Milligan, 11 Palmerston Place,	1885
	Sir Arthur Mitchell, 34 Drummond Place,	1859
	Dr Moir, 52 Castle Street,	1836
170	Dr Alexander Moir, 30 Buccleuch Place,	1876
	Dr Albert Edward Morison, Hartlepool,	1889
	Dr J. Rutherford Morison, 14 Saville Row, Newcastle-on-Tyne,	1882
	Dr John Mowat, 1 Hope Park Terrace,	1885
	Dr Claud Muirhead, 30 Charlotte Square,	1866
175	Dr R. Milne Murray, 10 Hope Street,	1884
	Dr A. D. Leith Napier, 67 Grosvenor Street, Grosvenor Square, London, W.,	1879
	Dr T. Goodall Nasmyth, Cowdenbeath, Fife,	1884
	Dr E. F. Neve, Dispensary, Srinagar, Kashmir, N.W. India,	1884
	Dr H. Newcombe, 5 Dalrymple Crescent,	1883
180	Dr P. Orphoot, 113 George Street,	1865
	Dr T. W. Pairman, H. M. Prison, Lyttelton, New Zealand,	1884
	Dr Paterson, 4 Coates Crescent,	1847
	Dr G. Keppie Paterson, 17 Forth Street,	1889
	Dr D. Noël Paton, 4 Walker Street.	1885
185	Dr Peddie, 15 Rutland Street,	1842
	Dr J. A. Philip, Rue Victor Hugo, Boulogne-Sur-Mer,	1878
	Dr R. W. Philip, 4 Melville Crescent,	1883
	Dr Playfair, 5 Melville Crescent,	1874
	Dr Edmund Price, 28 Mayfield Road,	1889
190	Dr J. H. Pringle, 5 Livingstone Place,	1886
	Dr T. Proudfoot, 13 Lauriston Place,	1889
	Dr S. Hale Puckle, Bishop Castle, Shropshire,	1885
	Dr Rattray, Portobello,	1874
	Dr William Richardson, Bath Lodge, Reading,	1884
195	Dr James Ritchie, 14 Charlotte Square,	1873
	Dr R. Peel Ritchie, 1 Melville Crescent,	1862
	Dr Argyll Robertson, 18 Charlotte Square,	1861

		Date of Admission.
	Dr G. M. Robertson, Morningside Royal Asylum,	1890
	Dr Ronaldson, 3 Bruntsfield Terrace,	1877
200	Dr J. Maxwell Ross, Calderbank, Maxwelltown, Dumfries,	1882
	Dr Joseph C. Ross, 98 Mosley Street, Manchester,	1884
	Dr Home Ross, 40 York Place,	1888
	Dr S. Rumboll, Belgaum House, Woodhouse Lane, Leeds,	1887
	Thomas Russell, Esq., 27A Westmuir St., Parkhead, Glasgow,	1888
205	Dr William Russell, 46 Albany Street, *Secretary*,	1884
	Professor Rutherford, 14 Douglas Crescent,	1866
	Dr F. A. Saunders, Denburn, Crail,	1884
	Dr Thomas R. Scott, Musselburgh,	1884
	Dr Andrew Semple, 10 Forres Street,	1883
210	Dr John Shand, 34 Albany Street,	1878
	C. H. E. Sheaf, Esq., Toowoomba, Queensland, Australia,	1871
	Dr G. F. Shiels, 229 Geary Street, San Francisco,	1887
	Dr W. H. Shirreff, Melbourne, Australia,	1883
	Dr J. Sibbald, 3 St Margaret's Road,	1859
215	Professor Simpson, 52 Queen Street, *President*,	1859
	Dr A. T. Sloan, 22 Forth Street,	1885
	Dr Andrew Smart, 20 Charlotte Square,	1865
	Dr G. D. Smith, 148 Ferry Road,	1877
	Dr John Smith, 11 Wemyss Place,	1856
220	Dr John Smith, Brycehall, Kirkcaldy,	1889
	Dr William Smith, Almond Bank, Midcalder,	1890
	Professor Grainger Stewart, 19 Charlotte Square,	1861
	Dr J. S. Stewart, 16 Merchiston Terrace,	1885
	Dr W. Stewart, Kirkwall,	1879
225	Dr William Stewart, 146 Ferry Road, Leith,	1889
	Dr H. J. Stiles, 5 Castle Terrace,	1889
	Dr S. Stirling, 6 Clifton Terrace,	1891
	Dr R. Stockman, 12 Hope Street,	1891
	Dr John Strachan, Dollar,	1867
230	Dr J. F. Sturrock, Homewood, Broughty Ferry,	1887
	Dr Allan C. Sym, 144 Morningside Road,	1889
	Dr William G. Sym, 58 Queen Street,	1889
	Dr Johnson Symington, 2 Greenhill Park,	1878
	Dr W. Taylor, 12 Melville Street,	1871
235	Dr C. C. Teacher, 16 Newington Road,	1887
	Dr C. H. Thatcher, 13 Albany Street,	1876
	Dr R. Thin, 6 Albany Street,	1890
	Dr Alexander Thom, Crieff,	1884
	Dr Alexis Thomson, 2 Coates Crescent,	1887
240	Dr John Thomson, 14 Coates Crescent,	1887
	Dr J. Stitt Thomson, Dalkeith,	1877
	Dr Francis Troup, 1 Minto Street, *Treasurer*,	1883
	Dr Batty Tuke, 20 Charlotte Square,	1864
	Dr J. Batty Tuke, jr., Balgreen, Murrayfield,	1886
245	Dr Dawson F. D. Turner, 7 George Square,	1890
	Professor Sir William Turner, 6 Eton Terrace,	1858
	Dr R. S. Turner, Keith,	1867
	Dr Underhill, 8 Coates Crescent,	1872
	Dr T. Edgar Underhill, Broomsgrove, Worcestershire,	1885
250	Dr D. Wallace, 66 Northumberland Street,	1887
	Dr P. H. Watson, 16 Charlotte Square,	1856
	Dr W. Watson, 34 Fountainhall Road,	1862
	Dr A. D. Webster, 20 Newington Road,	1883
	Dr A. B. Whitton, Aberchirder,	1886
255	Dr J. Lockhart Wilson, Duns,	1888
	J. L. Wilson, Esq., 4 Buccleuch Place,	1883
	Dr T. D. Wilson, 10 Newington Road,	1880
	Dr Oswald G. Wood, India,	1886
	Dr Russell E. Wood, 9 Darnaway Street,	1883
260	Dr G. Sims Woodhead, Beverley, Nightingale Lane, Balham, London,	1883

		Date of Admission.
	Dr Strethill Wright, 107 Chatham Street, Liverpool,	1871
	Dr Hamilton Wylie, 1 George Place,	1883
	Dr John Wyllie, 1 Melville Street,	1868
	Dr James Young, 14 Ainslie Place,	1859
265	Dr P. A. Young, 25 Manor Place,	1870

NON-RESIDENT.

	Dr J. J. Bailey, *Marple, Cheshire*,	1874
	Dr F. W. Barry, *London*,	1878
	Dr Bethune, *Toronto*,	1861
	Dr J. S. Beveridge, *Edenbridge*,	1861
270	Dr J. W. Black, *London*,	1865
	Dr Brodie, *Canterbury*,	1865
	Dr John Brown, *Burnley*,	1878
	Professor Cleland, *Glasgow*,	1864
	Dr Coghill, *Ventnor*,	1870
275	Dr P. M. Deas, *Exeter*,	1868
	Dr J. W. Eastwood, *Darlington*,	1871
	Dr R. B. Finlay, Q.C., M.P., *Middle Temple, London.*,	1864
	Dr Foulis, *Cupar-Fife*,	1859
	Professor Gamgee, *St-Leonards-on-Sea*,	1863
280	Dr Groesbeck, *Cincinnati*,	1875
	Dr Archibald Hall, *Montreal*,	1853
	Professor D. J. Hamilton, *Aberdeen University*,	1876
	Dr J. H. Hay, *Alloa*,	1880
	Dr Stanley Haynes, *Malvern*,	1864
285	Dr J. S. Howden, *Montrose*,	1856
	Dr James Johnston, *London*,	1871
	Dr George Keith, *Currie*,	1845
	Dr Lowe, *Lincoln*,	1845
	Dr F. R. Macdonald, *Inveraray*,	1860
290	Dr K. N. Macdonald, *Skye*,	1880
	Dr John A. Macdougall, *France*,	1875
	Dr John Mackay, *Aberfeldy*,	1881
	Professor M'Kendrick, *Glasgow*,	1870
	Dr W. O. Mackenzie, D.I.G.H., *London*,	1845
295	Dr T. J. Maclagan, *London*,	1875
	Dr J. Moolman, *Cape of Good Hope*,	1877
	Dr J. Ivor Murray, *Scarboro'*,	1857
	Dr Andrew Myrtle, *Harrogate*,	1859
	Professor Bell Pettigrew, *St Andrews*,	1873
300	Dr Phillippo, *Kingston, Jamaica*,	1860
	Professor W. S. Playfair, *London*,	1857
	Dr Priestley, *London*,	1854
	Dr Sinclair, *London*,	1850
	Dr Francis Skae, *Lerwick*,	1864
305	Dr T. Skinner, *London*,	1856
	Dr Van Someren, *Redhill, Surrey*,	1845
	Dr Somerville, *Galashiels*,	1877
	Dr Graham Steell, *Manchester*,	1877
	Professor Stephenson, *Aberdeen*,	1861
310	Dr H. R. Storer, *Newport, Rhode Island, U.S.*,	1855
	Professor Lawson Tait, LL.D., *Birmingham*,	1870
	Dr Thin, *London*,	1861
	Dr Turnour, *Denbigh*,	1843
	Dr Yellowlees, *Gartnavel Asylum, Glasgow*,	1862
315	Professor John Young, *Glasgow*,	1860

N.B.—*Members are requested to communicate with the Secretaries if they discover any errors or omissions in the List, and also to intimate all changes in their addresses.*

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TRANSACTIONS

OF

THE MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH,

FOR SESSION LXX., 1890-91.

Meeting I.—November 5, 1890.

Dr Clouston, *Vice-President, in the Chair.*

I. Election of Office-Bearers.

The following gentlemen were elected Office-Bearers for the ensuing session:—*President*, Professor Alexander R. Simpson; *Vice-Presidents*, Mr A. G. Miller, Dr Brakenridge, Dr P. H. Maclaren; *Councillors*, Dr John Smith, Dr Strachan, Dr Alex. Bruce, Dr George Hunter, Dr David Menzies, Dr J. H. Balfour, Dr R. M'Kenzie Johnston, Dr R. H. Blaikie; *Treasurer*, Dr Francis Troup; *Secretaries*, Mr F. M. Caird, 21 Rutland Street, and Dr William Russell, 46 Albany Street; *Editor of Transactions*, Dr William Craig, 71 Bruntsfield Place.

II. Election of Ordinary Members.

The following gentlemen were elected Ordinary Members of the Society:—William Smith, L.R.C.P. & S. Ed., L.F.P.S. Glasgow; Robert A. Fleming, M.A., M.B., C.M. Ed.

III. Exhibition of Patients.

1. *Mr A. G. Miller* showed—(1.) A young man who had suffered from thyroiditis—A. A., æt. 20. On the morning of July 24th patient

noticed that his neck was swollen. The swelling was red, soft, and painless. Patient did not know of any cause. The swelling was similar in shape to the thyroid gland, and rose and fell with the trachea in deglutition. It extended up to the thyroid cartilage and down to the sternum. Ice was applied to the neck and kept on for three days, at the end of which period the swelling had almost disappeared. On the fifth day patient was discharged, with the thyroid only slightly enlarged. Temperature on admission was 99° F. It fell to subnormal whenever the ice was applied, and remained down. Patient when shown to the Society had still an enlargement of the thyroid. In fact, it was much the same as when the patient had been discharged from hospital nearly four months previously. Mr Miller could only explain the sudden appearance and rapid subsidence of the swelling on the supposition that there had been an inflammation of the gland, probably from exposure to cold. The condition was very rare. He had not met with a similar case before. (2.) PATIENT, under care of Dr James Young. Man, æt. about 50. Upwards of twenty years ago patient strained his neck when carrying a table on his head, his head being thrown forcibly backwards. Since that time the thyroid gland has been enlarged. It began to grow more rapidly during the last three years. During the last six months the tumour has grown very rapidly, and has interfered with breathing. The tumour occupies the whole of the right side of the neck anteriorly, and extends partly over to the left side also. It is soft mostly, and at some parts apparently fluctuating. The trachea is pressed over to the left side considerably. The glands of the neck are enlarged. Mr A. G. Miller, at the request of Dr Young, demonstrated the case to the Society. He considered the traumatic origin of the bronchocele as the most interesting feature of the case. The sudden increase of the tumour after so many years of slow growth, along with the other features of the case, showed that it was now malignant.

2. *Dr John Thomson* showed a little girl, aged 2 years, suffering from PEDICULOSIS OF THE EYELASHES. She had no pediculi of the head or body, and those on the lashes were pediculi pubes.

IV. EXHIBITION OF INSTRUMENT.

Mr A. G. Miller showed a HOLLOW-HANDLED VOLKMANN'S SPOON, recommended by Dr De Forest Willard of Philadelphia. It was an improvement on Mr Barker's instrument, inasmuch as it did not require any special arrangement for shutting off the flow of lotion. A syphon apparatus was attached to the handle, and the flow was easily stopped by the pressure of the hand in working the instrument. The flow of lotion into the hollow of the spoon kept it clear of detritus, and also washed out the deep parts

of the wound thoroughly without the operator having to withdraw the spoon for that purpose.

V. Exhibition of Specimens.

1. *Dr Alexis Thomson* showed a specimen of OVERGROWTH OF THE SKULL FOLLOWING THE DISTRIBUTION OF THE FIFTH NERVE, and gave the following description :—

The rarity of the condition which this skull presents is indicated by the fact that there is only one similar specimen known to the writer—namely, one in the possession of Mr Jonathan Hutchinson, F.R.S. Its pathological interest lies in the fact that it furnishes a distinct addition to our knowledge of the influence which the nervous system may exert on the nutrition and growth of the different parts of the skeleton. The specimen was given to the writer by his brother, Dr D. G. Thomson, Superintendent of the Norfolk County Asylum, of which Institution the patient from whom the specimen was obtained was for many years an inmate.

The most striking feature which the skull presents (*vide* Plates) is a condition of asymmetry, and this depends upon a very considerable enlargement of the bones on the left side. Those on the right do not differ in any way from the normal. The enlargement affects all the bones of the left side, both facial and cranial, and stops very abruptly where the right and left halves of the skull meet along the middle line. The condition is one of hypertrophy of the affected bones, for they are enlarged in every dimension ; this is more marked in some bones than in others; it is very evident in the cranial vault, of which the left half is more convex, prominent, or bulging than the right ; also in the superior maxilla, of which the vertical height on the left side is 60 mm. compared with 40 mm. on the right ; again, in the lower jaw, in which the vertical height of the body is 36 mm. on the left half, and only 18 mm. on the right ; at the base of the skull the vertical height of the left external pterygoid plate of the sphenoid measures 42 mm., while the right plate measures 29 mm. These caliper measurements merely record what is evident at once to the unaided eye.

Added to this pure hypertrophy of the affected bones there is a condition of nodular and bossy thickening of their surfaces, which depends upon the formation of an amount of new bone by an over-active periosteum, and may be indicated by the term hyperostosis. This is specially evident over the zygoma and subcutaneous surface of the malar, both of which present a nodular, bumpy thickening, the surface of the new bone being minutely grooved and perforated by the vascular periosteum which covered it during life. These thickenings are identical in structure with the nodes met with on the surface of the long bones in syphilis. The alveolar border of the left superior maxilla shows this condition of hyperostosis in a still more exaggerated form ; the

edentulous margin of the jaw projects into the buccal cavity in the form of an elongated tumour-like mass with a minutely nodulated surface.

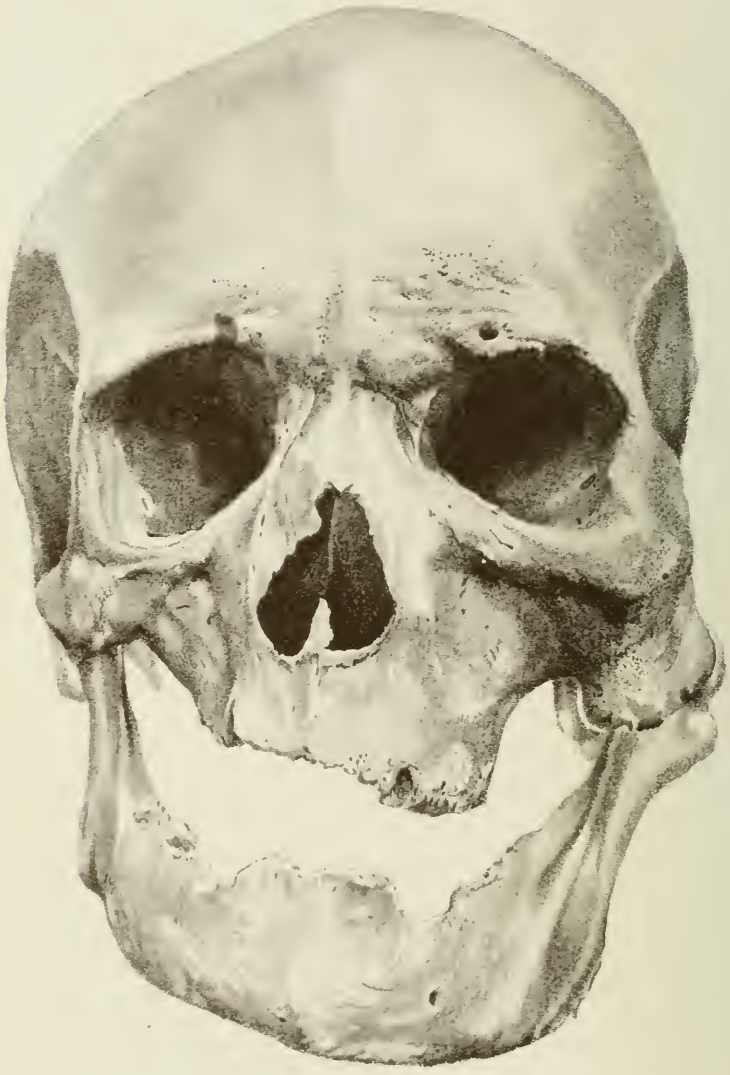
The condyle of the inferior maxilla on the left side is enormously enlarged, especially in its transverse diameter; it not only fills the glenoid cavity, which is correspondingly enlarged, but it has also come into contact with the outer aspect of the styloid process near its base, and a smooth circular facet has formed in the inner margin of the condyle where the latter has rubbed against the styloid.

Here and there the bony thickenings have assumed a distinctly tumour-like form, giving rise to exostoses, of which the largest grows from the inner table of the frontal bone, and projects into the cranial cavity. The tumour shows the cancellous structure characteristic of bony growths in this situation, while the surface in contact with the dura is dense or compact, and very uneven, from the presence of numerous hemispherical hillocks of various size, separated from each other by deep radiating fissures. It is curious that, although the tumour surrounds the frontal sinus, the cavity of the latter is very considerably dilated, so that its anterior wall bulges externally on the face, and its floor occupies a large portion of the roof of the orbit, from the cavity of which it is only separated by a thin translucent plate of bone.

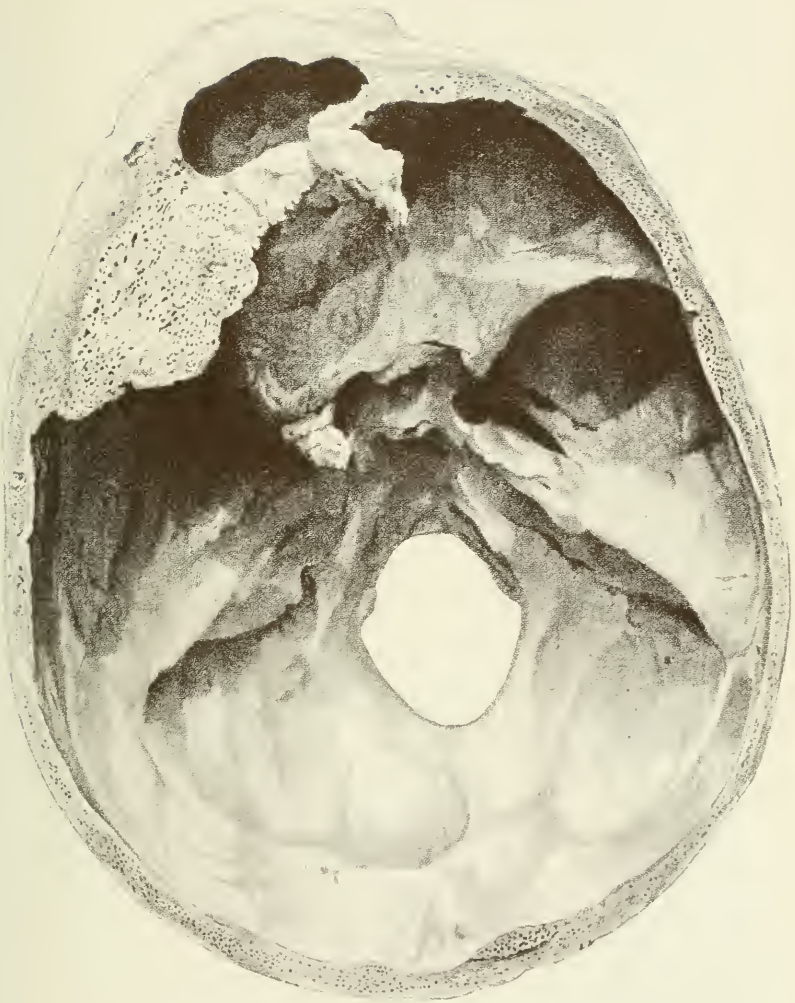
On the inner surface of the left parietal there are several scattered exostoses, varying in size from that of a pea to that of a cherry. There is, further, a conical exostosis springing from the left anterior clinoid process, which projects directly backwards so as to overhang the junction of the petrous temporal with the basi-occipital.

Associated with these various forms of overgrowth affecting the bones on the left side of the skull there is found a very pronounced enlargement of the grooves, canals, and foramina for the nerves and bloodvessels on the same side, more especially of those which serve for the transmission of the different branches of the fifth nerve, viz., the foramina rotundum and ovale at the base of the skull, and to an equal degree the supra-orbital, infra-orbital, malar, inferior dental, and mental foramina in the bones of the face.

The enlargement of these foramina was undoubtedly associated with a corresponding enlargement of the nerves passing through them. This fact being noted, it was then observed that the bony alterations which I have described followed with the greatest accuracy the area of distribution of the fifth nerve. It was found that the characteristic overgrowth and tumour formation affected every single bone in the anterior two-thirds of the skull on the left side, on their periosteal, pericranial, and dural aspects, but that these changes were not present behind the mastoid; what appeared at first sight to be a participation in these changes on the part of the left occipital portion, was found to depend rather upon an



Wm CATHIE LITH



Wm CATHIE LITH

alteration in the form of the latter, resulting from its constituting the connecting medium between the enlarged anterior portion of the left side and the unaltered occipital portion on the right.

This association of bony overgrowth in the area of distribution of a nerve, with an enlargement of its various branches, naturally suggests altered nerve influence as the cause of the overgrowth, and one would therefore appear to be justified in classing the morbid condition which the specimen presents with other so-called tropho-neuroses. Mr Jonathan Hutchinson, to whom the skull was sent for comparison with the one in his possession, with his characteristic scientific insight, was of opinion that the examination of one such specimen afforded sufficient grounds for the inference that there exist certain fibres in the fifth nerve which preside over the nutrition of the bones to which they are supplied, and which, when their function is exalted, may cause overgrowth and tumour formation throughout their area of distribution.[1]

So far as I am aware, there is no record of a similar result produced by aberration of trophic nerve influence in any part of the body ; there is abundant evidence to show that when the trophic fibres in the fifth are divided, or when their function is in abeyance, that the parts to which they are supplied may undergo atrophic and degenerative changes, and in a young subject may suffer from an arrest in their development. The affection known as hemiatrophia facialis illustrates these various results of loss of trophic nerve influence on the part of the branches of the fifth nerve in a very remarkable way ; in many respects, in fact, hemiatrophia is precisely the converse of the condition shown in our specimen—the former resulting from diminished, the latter from exalted energy on the part of the trophic fibres.

We have here, therefore, a distinct addition to our knowledge in regard to the influence of the nervous system on the development, growth, and maintenance of the different parts of the skeleton.

An aberration of this influence may be regarded as the essential factor in the production of the pathological changes met with in a considerable number of diseases. One group of these is characterized by the retarded or arrested development of the parts of the skeleton concerned, resulting from the trophic nerve influence on bone being in abeyance. Examples of this group are to be found in the arrested development of the bones of the limbs in infantile paralysis, and in the hemiplegias of early life, in the bones of the face in congenital wry-neck, and in hemiatrophia facialis. A second group is characterized by degenerative and atrophic changes in the bones, resulting from a perversion of the trophic nerve influence, of which examples are to be found in the fragility of the bones of the insane, in the articular disorganizations of locomotor ataxy, and probably also in the degenerative changes which go to make up the affections known as senile arthritis and malum senile of bone.

[1] *Inaugural Address, Roy. Med. Soc. Edin.*, 1890.

To these we are now in a position to add a third group, characterized by overgrowth and new formation of bone, resulting from an exaltation of the trophic nerve influence, examples of which are to be found in the specimens referred to in this paper, possibly also in the bony formations met with in arthritis deformans, and it may be in that rare form of bony overgrowth and tumour formation affecting the bones of the skull known as Leontiasis ossea.

The *clinical history* of the case from which our specimen was obtained may be stated briefly as follows:—[1]

The patient, a man aged 53, died about a year ago in the Norfolk County Asylum. His father was insane. The patient, a big, muscular man of 5 ft. 9 in. in height, had suffered from epilepsy major since infancy, but was able to go to school, learn to read and write, and subsequently learn his trade of shoemaker. After the age of 23 the fits began to tell on his mind, and he became of an irritable, hot tempered, and morose disposition, necessitating his removal to the workhouse infirmary of his parish, and while there he worked at his trade when able. About the age of 30, his irritability took a dangerous form; he would threaten to murder the officials, and he actually did injure some of the inmates who provoked him in a trifling way; he was therefore removed to the county asylum, where he remained till his death—23 years after his admission. His appearance was described as very peculiar, owing to the great enlargement of the left half of his face and head, otherwise he was well formed. He suffered from severe epileptic fits, occurring both night and day, generally in a cluster of three or four in succession about once a fortnight. The fits were of the grand mal type, sometimes preceded by an aura and attended with general convulsions. There was nothing peculiar about the fits. The hair, complexion, muscular movements, were precisely the same on both sides of the face. Sight and hearing were normal and equal on the two sides. Though the face was deformed, his expression was intelligent, and he could converse rationally; his articulation was slow and deliberate, without any defect whatever. His memory was good, he could read the newspaper, write a letter, and when well, used to work at his trade in the asylum workshop. Before and after his epileptic seizures he was very morose, sullen, and on the slightest provocation became abusive, threatening, and dangerously violent. At the age of 53, after an unusually exhausting series of fits, he never rallied, and died comatose in the status epilepticus.

When the body was examined, thirty-six hours after death, considerable difficulty was experienced in sawing through the skull cap on account of the large bony tumour growing from the left frontal bone. The membranes of the brain were healthy, with the exception of a disc-like plate of bone, 5 cm. in diameter, which

[1] For these notes I am indebted to Dr D. G. Thomson of the Norfolk County Asylum.

lay in the substance of the falx cerebri. The entire brain weighed 50 oz., of which the left hemisphere contributed 16 and the right 25 oz. The convolutions on the anterior and superior aspect of the frontal lobe on the left side were displaced, atrophied, and flattened by the large bony tumour already referred to. Nothing further was observed as abnormal in the body with the exception of a disc-like plate of bone, about 5 cm. in diameter, lying in the substance of the central tendon of the diaphragm.

It may be inferred from the above notes, that the bony overgrowth, of which the patient was the subject, commenced in early life, how soon it is impossible to say, for the very insidious progress of the malady prevented the patient from noticing at what period the deformity first became evident. Apart from the deformity, the facial part of the affection does not appear to have caused any symptom whatever. In the cranial bones, on the other hand, the bony tumours which projected from them and pressed upon the brain caused symptoms, among which may be mentioned the epileptic seizures from which the patient suffered during the greater part of his life, these being probably due to the pressure of the large frontal enostosis to which reference has been made.

2. *Dr Kenneth M. Douglas* showed a SPECIMEN which Dr P. H. Maclaren had removed on the preceding Friday by abdominal section—Dr Maclaren, owing to slight indisposition, being unfortunately unable to be present. The history of the case was briefly as follows:—The patient, a healthy labourer, aged 24, went to work as usual on Friday, October 24, and had a healthy action of the bowels before leaving home. Two hours later he began to experience "twisting" pains in the umbilical region, which became so bad that at 10.30 he had to go home. During the day the bowels acted more than once, and he vomited greenish semi-solid stuff. In the afternoon he was seen by a medical man, who ordered some "pill" and an injection. These acted fully, and on the next day two further injections were ordered. The vomiting and pain persisted despite all treatment, and on Wednesday, Oct. 29, he was advised to come to Hospital. So far as could be discovered, none of the stools contained blood, nor an excessive amount of mucus. On admission, the man was found to be collapsed and depressed; temperature, 97°. The abdomen was moderately distended, the intestinal coils being evident through the walls; there was no pain nor tenderness, no perceptible tumour, nor definite resistance, nor dulness on percussion. As the case did not appear to be one of absolute intestinal obstruction, small doses of calomel were ordered, and no food beyond iced milk; turpentine stupes were also applied. Next day, further careful examination disclosed no tenderness nor tumour; the abdominal walls were quite soft and relaxed. Since admission, the bowels had acted twice, and the stools were described as healthy. The

treatment was altered, and the patient brought under the influence of morphia. On the following day (October 31) the patient's state was changed. He was flushed and restless, the temperature was 100°·4, and the pulse 144, respiration being only 11 per minute. The abdominal walls were tense and hard. After consultation with Mr Duncan, it was agreed to operate, peritonitis having obviously supervened. The abdomen was therefore opened in the middle line from the pubes to the umbilicus, and at once some clear fluid escaped. The lesser bowel was distended and of a dark red colour. After some search, which entailed considerable eventration, the mischief was discovered, and this piece of bowel upon the plate was drawn out of the wound. At first sight it appeared as if a fleshy band of the thickness of the little finger encircled the bowel, but this proved to be the upper limit of an intussusception, at or near the ileo-colic valve. The state of the gut necessitated its excision, and the operation was completed by stitching the two ends of the divided bowel in the skin wound. The patient died some four hours later. The outer tube of the intussusception had sloughed, so that at one point the mucous surface of the middle tube was exposed; one could also clearly see the bowel above the intussusception, the portion of cæcum resected, and the lower end of the intussusception passing into it.

VI. Original Communications.

1. WRY-NECK: ITS VARIETIES AND THEIR TREATMENT.

By Thomas Annandale, F.R.C.S. Ed., Regius Professor of Clinical Surgery, University, Edinburgh.

As the result of considerable experience in observing and treating cases of wry-neck, I would suggest the following classification of the varieties of this deformity, and I offer some remarks upon their treatment.

1. Simple.
2. Spasmodic (Temporary).
3. Spasmodic (Persistent).
4. Complicated.

1. *Simple.*—This is the result of contraction of one sterno-mastoid muscle, which may appear as a contraction of the whole muscle or of only its tendinous portion. It is met with in every degree of aggravation, and its *causes* may be—

a. Congenital.

b. Injury to one muscle during child-birth, or from accident or surgical operation.

c. Paralysis or weakening of the muscle upon one side.

d. Bad habit of holding the head and neck to one side, as in glandular and other painful affections.

I have at present under my care a woman, 25 years of age, who has a well-marked and uncomplicated contraction of the right sterno-mastoid causing wry-neck, the result of suppurating cervical glands. There has been no loss of substance of the muscle, nor is there any contraction of the soft parts, but the patient states that for several years, during the progress of the gland affection, she kept her head turned to the right, and this habit has caused the muscular contraction.

A few years ago I met with an interesting case of contraction of one sterno-mastoid the result of imitation. The patient was a young boy belonging to a neurotic family, and the condition of wry-neck commenced by his imitating an elder brother who suffered from wry-neck, and whose sterno-mastoid I divided with a perfect result. Means were used to prevent the imitation, but they were unsuccessful, and the deformity became permanent in the second brother, who also was only cured by the division of his sterno-mastoid.

Treatment.—Stimulate the weakened or paralyzed muscle by proper massage, galvanism, counter-irritation, and attention to health; and if these means fail, divide the contracted muscle. If the contraction affects the tendinous portion only, the division of this portion is usually sufficient.

2. *Spasmodic (Temporary).*—In these cases the spasmodic contraction of the one sterno-mastoid muscle is not constant, and other muscles of the neck may be similarly affected.

Causes.—Some temporary derangement of the general nervous system, or some local nerve or muscular irritation. This condition may become more permanent if not relieved. I have seen cases in which temporary local nerve irritation in connexion with inflammation of the cervical vertebræ or other cervical tissues has caused spasmodic contraction of one sterno-mastoid, and this contraction has become persistent owing to the difficulty of overcoming the acquired habit of holding the head to one side.

Treatment.—Careful attention to the condition of the general nervous system, the removal of any probable cause of local irritation if it can be discovered, and properly applied massage.

3. *Spasmodic (Persistent).*—This variety may depend upon a more or less continuous spasmodic contraction of one sterno-mastoid muscle only, but not unfrequently other muscles in the neck are also affected. In some of these cases the condition is most distressing, the head and neck being constantly jerked to one side, or jerked backwards, the patient not having any control over such movements. It may be possible for the patient to steady the head for a short time, but on attempting to turn it, it starts off again, and the uncontrollable jerking continues. Cases of this kind vary as regards the amount of spasmodic movements and their frequency.

Causes.—May result from the more temporary cases, or from special nerve irritation, but the most common cause which I have met with is the result of constant and long-continued movements of the head and neck to one side, such as are required in certain occupations,—in the case of compositors and workers at certain machines, for instance.

Treatment.—This class of case does not, as a rule, become cured by any general medical, local, or mechanical treatment; but the division or excision of a small portion of the spinal accessory nerve high up in the neck is followed by good results. On the 7th of February 1878—following the example of M. de Morgan (see *British and Foreign Med. Chir. Review*, July 1866)—I operated upon a case of this kind by dividing the spinal accessory nerve. This case, which was a perfect success, was published in the *Lancet* for April 19th, 1879. Since then, I have operated with the same good result upon a second case; and Dr Cotterill has similarly treated, with an equally good result, a third case in my wards. In all these three cases the condition was the result of the patient's occupation. In one, the patient worked at a power-loom machine, and the other two patients were compositors.

It is well here to note that after the division of the spinal accessory nerve the spasmodic movements do not always cease at once, but in some cases the muscular contractions and irritability remain for a time before the case is perfectly cured.

Mr Southam (*British Medical Journal*, July 11th, 1885, and January 31st, 1891) gives an account of several successful cases of this deformity treated by the excision of a portion of the spinal accessory nerve; and Mr Collier (*Lancet*, June 21st, 1890) reports a successful case in which he passed round the same nerve a wire ligature slightly compressing the nerve. I think that the division of the nerve, taking care to separate the divided ends, or the excision of a portion of it, have given the best results, and that therefore one of these methods should be adopted in preference to merely stretching or compressing the nerve. I am also of opinion that the best incision to reach the nerve is one made along the upper and anterior border of the sterno-mastoid muscle.

Aggravated cases of this spasmodic variety are met with in which the posterior cervical muscles are also affected. In one such case, after the division of both spinal accessory nerves had failed to give relief, I, by subcutaneous incision, divided all the attachments of these muscles to the occiput. The relief was, however, only temporary, and a better proceeding in these cases will probably be to divide or excise portions of the posterior branches of certain of the cervical nerves. Mr Noble Smith in a letter to the *Lancet*, June 28th, 1890, refers to a case in which he, after excising a portion of the spinal accessory nerve, subsequently for spasm of the rotators on the opposite side excised portions of the posterior branches of "some of the cervical nerves." The result was successful.

My friend Professor Keen of Philadelphia has recently made a careful study of the question of division of the cervical nerves in this deformity, and he has published an important paper (see *Annals of Surgery*, January 1891), giving rules for an operation which will enable the surgeon to divide the posterior divisions of the first three cervical nerves, so that the chief posterior cervical muscles which rotate the head may be acted upon and spasm of them relieved. Professor Keen's rules are as follows:—

"*First step.*—The field of operation having been shaved and disinfected, make a transverse incision about half-an-inch below the level of the lobule of the ear, from the middle line of the neck posteriorly, or even slightly overlapping the middle. This incision should be 2½ to 3 inches long.

"*Second step.*—Divide the trapezius transversely.

"*Third step.*—Dissect up to the trapezius and find the occipitalis major nerve as it emerges from the complexus and enters the trapezius. In the complexus is an intra-muscular aponeurosis. The nerve emerges from the complexus at a point between this aponeurosis and the middle line, usually about a half inch below the incision, but sometimes higher up, and then enters the trapezius. It is always a large nerve of the size of a stout piece of catgut, and it is easily found if sought for at the right place.

"*Fourth step.*—Divide the complexus transversely at the level of the nerve. This division should be made by repeated small cuts, so as not to cut the nerve which is our guide, after which dissect the nerve still further down from the anterior surface of the complexus, where it arises from the posterior division of the second cervical. Cut, or better, exsect a portion of the posterior division before the occipitalis major arises from it, so as to catch the filament to the inferior oblique muscle. This divides the *second cervical.*

"*Fifth step.*—Recognise the inferior oblique muscle by following the sub-occipital nerve towards the spine. The nerve passes immediately below the border of the muscle.

"*Sixth step.*—Recognise the sub-occipital triangle formed by the two oblique muscles and the rectus capitis posticus major. In this triangle lies the sub-occipital close to the occiput. It should be traced down to the spine itself, and be divided, or better, exsected. This divides the *first cervical.*

"*Seventh step.*—An inch lower down than the occipitalis major, and under the complexus, is the external branch of the posterior division of the third cervical to the splenius. When found, it is to be divided or exsected close to the bifurcation of the main trunk. This divides the *third cervical.*"

4. *Complicated.*—Included in this class are those cases in which one of the following conditions is present:—

a. Disease of the cervical vertebræ.

b. Cicatricial contractions from wounds, ulcerations, or other conditions causing destruction of tissue.

c. The presence of enlarged glands, or morbid growths, or other diseased conditions in the neck.

Treatment.—In this variety of wry-neck the particular complication may prevent the cure of the deformity, but when possible the complicating cause should first be relieved or removed, and then manipulative or operative means used to overcome any distortion which may remain.

When in any case of wry-neck tenotomy of the muscle has been performed, massage and movements of the head and neck should be carefully carried out when the wound has healed and the muscle has united. Except in very aggravated cases, special splints or other appliances are not required after such an operation.

Mr Caird expressed his great pleasure at hearing a paper which gave the result of such extensive experience. He would like to ask Professor Annandale if, in the cases of continuous spasm of the sterno-mastoid, there had been any hypertrophy of the muscle or exaggerated curvature or change observed in the clavicle.

Mr Cotterill remarked that in criticising the two operations proposed for division of the spinal accessory nerve, he was strongly of opinion that the better operation was that which sought for the nerve by the upper incision, viz., one along the anterior border of the sterno-mastoid muscle, three inches in length, commencing at the apex of the mastoid process. The objections to the lower incision, as usually recommended, at the posterior border of the muscle, were:—(1.) That the position of the nerve was variable. (2.) That it might in this position be hard to distinguish it from other nerves, viz., the third cervical, small occipital, and great auricular. (3.) That when it was found it was then necessary to dissect it up some distance before the point at which it gave off its branches to the sterno-mastoid muscle were reached. Mr Cotterill, in referring to a successful case brought before the Society, pointed out that after section of the nerve the patient might have occasional relapses for some months; but that in his case there had now been for over a year no return of spasm, though the patient had returned to his work as a compositor, which occupation had brought on the disease.

Dr P. A. Young rose to mention a remarkably rare cause of wry-neck which had recently come under his notice. A girl, aged 7 years, was brought to him with reference to the advisability of using massage for wry-neck. The arms were fixed to the sides on account of an osseous degeneration of the muscles passing from the trunk to the arms. The sterno-mastoid on the right side was much affected, causing wry-neck, and the muscle on the left side was beginning to be affected. This interesting and rare disease had been called by Münchmeyer, *Myositis ossificans multiplex progressiva.*

Henoch with his large experience had only seen one case. He merely mentioned this case now, as it exemplified a rare cause of wry-neck, and hoped at a future meeting to bring the case more fully before the Society.

Dr Symington wished to know if Professor Annandale exposed the internal jugular vein in the course of the operation upon the spinal accessory nerve at the anterior border of the sterno-mastoid. He thought it well for surgeons to remember that the nerve sometimes passed behind the vein, instead of in front of it. He ventured to suggest the propriety of attempting to divide some of the upper spinal nerves in those cases in which several muscles of the neck were affected.

Dr Burn Murdoch thought that the thanks of the Society were due to Professor Annandale for having so clearly brought before it the result of treatment of spasmodic and non-spasmodic wry-neck. He related the case of a child who suffered from congenital non-spasmodic wry-neck, with marked secondary lateral curvature of the spine, where division of the sternal and clavicular attachments of the contracted sterno-mastoid was rapidly followed by the head becoming erect, and under gymnastic exercises the lateral curvature of the spine soon disappeared.

Prof. Annandale was always glad to have the criticisms of his anatomic colleagues. He was glad that Mr Symington approved of the incision along the anterior border of the sterno-mastoid. He agreed that there might be a difficulty in finding the nerve, but he suggested that if there was any difficulty the branches which passed to the sterno-mastoid might be traced upwards so as to reach the main trunk. He quite thought that in cases in which other cervical muscles were involved, a division of the nerves supplying them might be useful. He wished to emphasize a remark by Dr Cotterill, that the good result of division of the spinal accessory nerve was not always immediate, and this he attributed to the muscular irritability continuing for some little time. In answer to Dr Caird, he thought division of the nerve with separation of the ends sufficient, and also that he had not noticed alteration in the clavicle in spasmodic cases. He considered Dr Young's case a most interesting one. He had seen ossification of certain muscles, but he had never seen this condition in the sterno-mastoid.

2. DERMATITIS HERPETIFORMIS: A CLINICAL STUDY.

By W. Allan Jamieson, M.D., F.R.C.P. Edin., Extra Physician for Diseases of the Skin, Edinburgh Royal Infirmary; Consulting Physician, Edinburgh City Hospital.

It is matter of common experience to the resident in any large city, with what comparative frequency he seems to meet one to whom he has been recently introduced, also a town dweller, but

hitherto unknown. He has now learned to note and recognise him in the crowd of passers-by, and he finds himself astonished that a face become at once familiar had been beforetime unobserved. The same thing occurs in Medicine. Some one more watchful than his compeers isolates, from what has appeared a tolerably harmonious group, a class of cases possessing certain features in common, describes them afresh, and attaches to them a distinctive name, which at once invites popular attention. Should his conception prove correct, additional and confirmatory evidence is soon forthcoming, the position of the new disease becomes established, and fresh light is shed on much which was previously obscure.

It is thus with the disease, which almost certainly includes several varieties named by Duhring Dermatitis herpetiformis, but which some with much less reason would prefer to call Hydroa. Bazin undoubtedly recognised the complaint, and gave a somewhat precise description of three forms, yet his observations must have been founded on the examination of an insufficient number of instances, since one of the best marked of his types has been discarded, while another has till recently been denied a place. Thus his vesicular hydroa is now viewed as identical with herpes iris, a variety of erythema multiforme, and his hydroa vacciniforme is not admitted as a definite known disease either by Liveing or Crocker. Tilbury Fox also was acquainted with the class of cases under consideration, but like Bazin he somehow missed the opportunity of making an effective picture, yet some examples were identified from his description. Duhring was more fortunate, and we must add more persevering in the face of many difficulties and a good deal of adverse criticism and actual incredulity. He repeated time after time his propositions, published case after case, and thus succeeded in fixing attention. His views have gained novel aspects and assumed a wider scope, as a result of the masterly inductions of Brocq[1] and the trenchant writings of Unna. Little is now left save to fill in details, and thus to complete a chain already fairly continuous.

Mr Hutchinson has provided some materials for this in his lectures on varicella prurigo, and in his remarks on an eruption allied to xeroderma pigmentosum, or Kaposi's disease. In neither instance, however, though as interesting, ingenious, and suggestive, has he been quite as happy as usual, for by concentrating his gaze too exclusively on an occasional sequence of phenomena, or on the localization of the lesions, he has lost sight of a more important connecting link. The present writer is all the more alive to this, as he also, previous to and quite independently of Mr Hutchinson, fell into the same error with respect to one group of these cases, —those, namely, apparently related to xeroderma pigmentosum, misled by the same resemblance in features.

[1] *De la Dermatite Herpétiforme de Duhring.* Paris, G. Masson, 1888.

The definition formulated by Unna[1] is perhaps as good a one as is at present available in the existing state of our knowledge. Dermatitis herpetiformis is, according to this, "a chronic, sometimes an acute, neurosis of the skin, not markedly interfering with the general health. This causes a more or less universal eruption, associated with burning and itching sensations, and regularly recurring after intervals of complete or comparative immunity. The type is erythemato-bullous, which, however, may undergo considerable modification."

There are four features characteristic of the complaint: 1. The polymorphic nature of the eruption. 2. The paræsthesiæ which accompany it. 3. Its course, in the main chronic, exhibiting a decided tendency to relapse or recur. 4. The relatively good state of the general health. All these need careful consideration.

1. The polymorphic nature of the eruption. This is twofold. In each case there are two, sometimes more forms of primary or secondary eruption present at the same time; and again, the characters of the eruption may vary in successive outbreaks. The type has been defined as erythemato-bullous. Erythematous blotches or flat papules are always to be found at some, usually an early, stage, and as a rule make up the bulk of the lesions, but vesicles, bullæ, or pustules may one or all be met with in association with blotches or flat papules. The erythematous patches are in themselves commonly pretty well defined. Now and then they resemble the wheals in urticaria, and this peculiarity is occasionally specially marked in the case of children. In some instances the vesicles develop on the erythema or papule, in others the vesicle or bulla—which latter may be of large size—is marginal or quite distinct from the erythema. The primary pustular form is certainly the more rare, its occurrence may be due to the general health being much lowered, but it may also be explained, as Unna thinks, by the invasion of pyogenic organisms. The vesicles are not limited to the skin, they have been seen on the mucous membranes, as inside the mouth. One distinctive character must be noted, that the lesions are grouped, herpetiform, not isolated. The crusts or hard flat scabs which succeed the vesicles are peculiar. They are somewhat angular in shape, are hard, dry, and brown. In one variety the face, neck, hands, and exposed parts are primarily at least, and in most cases solely, attacked. In others the eruption shows a partiality for the neighbourhood of the articulations, and for covered portions of the body, but while it starts from these localities, it may extend widely. The form which is limited to exposed parts, as a rule commences in early childhood, while the other and more common of the varieties may begin at any period of life. The first mentioned form leaves scars which may be extensive, the ears in particular suffering, and the cicatrices so produced prone to contract and occasion disfigurement.

[1] *Monatshefte fur praktische Dermatologie*, 1st August 1889.

In the milder type blotches or stains, pigmentation, leucoderma, or even minute white glancing scars, represent the conditions seen in some persons on the subsidence of the more acute symptoms, or, when the eruption has vacated one part to attack another, such changes are left, temporarily at least, in its wake.

2. The paræsthesiæ which accompany it. Itching is complained of in all those cases where the covered parts of the body are affected. The itching is usually intense and distressing, but it is not constant, it is apt to become aggravated at times. Before, or concurrent with the eruption of a fresh crop of lesions, itching is a prominent symptom, but when the vesicles have formed or the papules have risen, the itching becomes less in degree, to awake again with the formation of another batch of blotches or the appearance of new blisters. In the form which attacks the face, ears, and hands, pain or burning sensations in these parts precede each exacerbation. Since in these cases the disease is usually quiescent during the winter months, there is then a period of freedom from subjective impressions, which, however, manifest themselves as the complaint reasserts itself in spring or early summer.

3. Its course, in the main chronic, exhibiting a decided tendency to relapse or recur. Unna regards this as the most important symptom of the disease, and until these features have manifested themselves unmistakably, he would reserve his diagnosis. This, however, necessitates the introduction of the element of time, and while such may render our opinion a more precise one, there are cases which can be identified before this has fully declared itself. Some authors, as Brocq, speak of an acute form. An example of this kind has been put on record by Hautecoeur,[1] but this refers more to the rapidity with which the lesions themselves evolve than to the absence of recurrences. Chronicity, indeed, may be conceded as essential, while fluctuations in intensity, relapses, or true recrudescences are parts of the ailment. Such may finally terminate or be indefinitely absent.

4. The relatively good state of the general health. Considering the extent of surface involved and the severe itching, rendering sleep unrefreshing, the maintenance of health of a fairly high standard is remarkable. Still, some patients complain of weakness—so much so, indeed, as to necessitate their taking to bed. There is sleeplessness and emaciation, or at least some loss of flesh. One symptom is pretty constant, though its cause is obscure, this is the occurrence of diarrhœa. More males than females are affected in the generalized form ; that localized to the face, hands, and exposed parts has so far only been seen in males. The duration is quite undetermined in the generalized form. The localized ceases or much lessens in severity about or before the twentieth year.

Many authors now include those cases which have been

[1] *Annales de Dermatologie et de Syphiligraphie*, 25th Janvier 1890.

described under the term herpes gestationis as a part of dermatitis herpetiformis, the circumstances of the patient modifying a little the features displayed. The eruption may appear either during pregnancy, usually not earlier than the tenth week, and may continue more or less well marked till delivery or even till lactation is established. In this case there is often a fresh outbreak or an intensification of the complaint shortly after delivery. Or it may not manifest itself till some days after the confinement, and may last for one, two, or even three months as a series of outbreaks, each of these being preceded, in nearly every instance, by itching sensations. It is liable to recur in successive pregnancies, but there are cases on record in which a mother has escaped an attack in some while she suffered in others. It is apt, however, to increase in intensity and extent at every fresh recurrence.

This form of dermatitis herpetiformis starts on the limbs, in particular on the hands and arms, though it occasionally first appears near the umbilicus. The lesions resemble those described, pustules are rare, unless the transformation of the vesicles into pus be regarded as such. The general health and the appetite remain good, even though, as Brocq remarks, those affected are fatigued or even prostrate. Sometimes the relish for food fails at the commencement of a seizure, but is regained though the complaint persist.

But this does not exhaust the relationships of this peculiar disorder. In 1872 Von Hebra described a pustular disease of the skin of much gravity, and which he looked on as allied to the herpes group. This in time came to be referred to under the name of impetigo herpetiformis. Only some fourteen or fifteen cases have been published, and all with one exception have so far ended fatally. It was at one time thought that it occurred exclusively in pregnant women, but Kaposi has related an instance in which it affected a man.[1] Small yellowish, superficial pustules appear, which begin as such and remain unchanged throughout their entire course. These are arranged in groups or circles on an erythematous base. The pustules tend to run together and to dry into yellowish, greenish, or brownish crusts, beneath which is a red, moist, excoriated surface. This heals without ulceration, leaving no cicatrix. At the periphery of these patches fresh groups or rings of pustules develop, and in this manner the disease spreads over wide areas. The inner sides of the thighs and the anterior surface of the body are the seats of the eruption at first and preferentially, but in process of time it may extend to other parts, or even invade the mucous membrane of the mouth or elsewhere. Each outburst is preceded by rigors, fever, and disturbance of the general system. The question of kinship has been carefully considered by Duhring,[2] and on perusal of his arguments one must, I think, go even further than he is quite inclined to, and

[1] *Viertelj. fur Derm. und Syph.*, 1887.
[2] *American Journ. of Med. Sciences*, March 1890.

conclude that such cases are best classed as examples of the pustular form of dermatitis herpetiformis. The main difference arises from the fact that such are pustular throughout, and that a species of septicæmic infection occurs. The pregnant or parturient state affords special facilities for the development of danger from this source. Sherwell has come to a similar conclusion from the consideration of a case which came under his own notice.[1]

Before proceeding to discuss the nature and diagnosis of the complaint, the relation of typical examples, illustrating as many varieties, may serve to render the picture already sketched more complete.

1. T. M., 23, stoker of a locomotive engine, came to see me on 21st October 1889. He was a healthy looking man with a good fresh colour. Nine months since he was much over-heated, then perspired profusely, and immediately after the present eruption appeared. This has since then never entirely left him, though at times pretty quiescent, again to suffer fresh exacerbations. It first showed itself on the chest, from thence it spread to the arms, back, thighs, buttocks, neck, face, and head. There was in the very early period not much itching, but this became intense later on. When the first outbreak occurred, he felt hot, sick, and vomited, and his appetite failed. Before any exacerbation he again was squeamish, and was compelled to go to bed for a couple of days. He did not regain his inclination for food till recently, and was weak. When seen he was much better, though there was no improvement in the condition of his skin. His tongue was clean, all the functions were normally performed, some constipation which persisted for a time had ceased. There was no history or the least presumptive evidence of syphilis. The localities affected were the neck moderately, and face slightly. The sides of the thorax markedly, the axillæ and margins of the axillæ, the clavicular and scapular regions. On the arms, while the deltoid region, over the elbows outside, and in the flexures inside were affected, the remainder of the arms and hands were free. The eruption notably occupied the lumbar and sacral region, extended across the abdomen, spread down the thighs, being prominent on the nates, and was seen in the ham; but the sternal region, that between the scapulæ, the legs elsewhere and the feet, were not invaded. In several other instances in men the very same portions of the body were those alone or most specially implicated.

The eruption consisted of the following elements:—1. Erythematous patches, varying from a pea to a shilling or larger, irregular in shape and distribution, but numerous. These were bright red in hue, and gave rise to a certain degree of infiltration of the skin. There were also red macules due to lesions which had formerly been existent but had disappeared. 2. Vesicles and small bullæ.

[1] *Journ. of Cut. and Genito-Urinary Dis.*, Dec. 1889.

These were tense and of a clear pale straw colour, with little tendency to rupture spontaneously. They were found in groups of three or four or more, were seated sometimes on unaltered skin, at others on an infiltrated base; some had, many had no areola. The largest were in the neighbourhood of the axillæ. They were in great measure placed marginally as regards the erythema. 3. Scabs and thin, firmly adherent, often angularly-shaped crusts, the results of former vesicles, partly, however, of scratching which had caused oozing. 4. Pigmentation, well marked near the axillæ and on the thorax, as deep brown staining, but present also on other parts, as the abdomen and groins, very noticeable over the sacrum and lower dorsal region. 5. Leucodermic patches or spots scattered here and there throughout the pigmented areas. 6. Some minute scars.

The itching was most troublesome at night, or when he became hot, and at such times was nearly unbearable. He was admitted into Ward 4, under the care of Dr MacGillivray, where he remained till the 29th November. By the kind permission of Dr MacGillivray I was able to direct the treatment. This consisted of nightly warm baths of starch and potassa sulphurata, followed by painting with a boracic calamine lotion, and the administration of arsenious acid in pill, the dose being at first one-fiftieth, later on one twenty-fifth of a grain, thrice a day.

In the end of November the itching had ceased, and no fresh bullæ had come out for some time. The skin was everywhere smooth, the pigmentation much less, the leucoderma less marked, while he felt quite well, so that he was discharged. He wrote to me twice subsequently to his return home on the 18th December. By the end of the year he was again nearly as bad as ever, though the eruption was confined to the upper part of the body.

On the following instance the multiformity of the lesions, and the manner in which one or other form came into prominence, is fairly well shown.

W. W., 24, miner of shale, Broxburn. A well-built and healthy looking man, who had no anæmic aspect, nor had at any time suffered from rheumatism. In the early part of January 1889, a patch of eruption appeared on the left hip and on the left arm over the tip of the elbow, both points where pressure was exercised when at work. The eruption extended and became localized in the same situations as in the case just described, except that the face escaped. When seen he had been taking iodide of potassium; this had at once aggravated the eruption, and increased the number and size of the blebs. The soft palate was congested, and there were some vesicles in that situation also. The lesions varied in type. At one time a group of blisters came out on a reddened base, which was also to some extent congested; the central vesicles of each group shrank, while new ones formed at the edge, finally a reddish blotch or discoloration remained. At

another time it was made up, especially over the abdomen, of dry erythematous areas, presenting a marginate aspect like tinea circinata or body ringworm. These were a little scaly and gyrate at the edges. The condition altered thus from month to month, no treatment doing much good. In June 1890 he had become thinner, the skin was dry, sallow, and pigmented, there were thickly set papular and erythematous lesions on the chest, elsewhere vesicles were present as well. He was admitted to Ward 29 under Dr Muirhead's care. His diet was restricted, for it was found he had been living pretty freely in the country, and this seemed at first to produce an improvement. Then he was ordered alkaline baths, followed by inunction with the unguentum sulphuris. He became nearly free from his complaint by the middle of July, and was discharged, but some fresh spots soon reappeared after he went home. In his case the itching was most intense when the eruption, and especially the vesicles, were coming out, less at other times. The pigmentation was observed to become intensified after he had taken arsenic in small doses for a very considerable length of time. This effect of arsenic has been noticed by Dr Handford and Mr Hutchinson among others.[1] He was last seen on the 30th of August 1890, when he still presented the same eruption and in the same localities, though he thought he had considerably benefited by sea-baths at Portobello, which he said diminished the itching markedly.

The following case has been seen at intervals for more than two years, and is now apparently cured :—

G. W., 73, upholsterer by trade, has been a total abstainer for fifty-two years, and when an apprentice only indulged very moderately in ale. He does not look his age, his hair and beard are iron gray, while he is active and intelligent. He was first seen in June 1888. He was in good health and made complaint of nothing save the eruption, the itching, and consequent sleeplessness. Two months ago some red blotches showed themselves on his left elbow, each extended at its margin, while it faded in the centre. Then similar erythematous patches came on the right elbow, at margin of the axillæ, over the sacrum and lower part of the loins, and finally on the shoulders. At first there was no more than erythema, in some situations an inch, in others less in breadth, but a month since large vesicles and bullæ formed on the advancing edges of the patches. On the loins especially the eruption extended widely. He was ordered ten grains of iodide of potassium thrice a day, and a calamine lotion containing a little perchloride of mercury locally. The medicine caused iodism, much pain, a great increase in the number of bullæ, some forming in the mouth, and had therefore to be discontinued in course of a few days. In October of the same year pustules appeared in place of bullæ and vesicles, but these ceased under the use of nitro-

[1] *Trans. Clin. Soc. of London*, 1888 ; *Archives of Surgery*, July 1890.

hydrochloric acid and strychnia. In December 1889 it is noted that while the same localities are still implicated, at present the blisters are limited to the scrotum, penis, and inside of the thighs, but are accompanied by great heat. There were now seen patches of pigmentation, and white glancing scars or spots, as well as erythema and crusts. For a time baths of potassa sulphurata and starch, when not taken very hot, relieved him, but eventually seemed to aggravate the eruption. In the middle of October 1890 he was quite well as regarded the eruption, all the discoverable remnants being some staining and red mottling of the skin in the localities previously so severely affected. He ascribed the cure to his having bathed himself with a moderately strong solution of borax in water at night, and with a weak solution of permanganate of potass in the morning.

When dermatitis herpetiformis attacks children the appearances vary somewhat from those seen in adults. The following case illustrates one form, at least, which the disease assumes in them :—

J. D., 2½ years, an only child, who looks pale and not very robust. Six months ago the present eruption appeared, and has been better and worse ever since. A small patch of erythema first comes out, and on or near this a vesicle forms, or a bulla, with clear straw coloured contents, this flattens down and partly becomes crusted over, or from scratching becomes excoriated, and finally only a stain remains. At times, however, there is left a white mark with pigmentation round it. The limbs are specially affected, as also the nates and lower portion of the loins, but there are numerous lesions on the back and abdomen. The grouping of the vesicles is not so distinct as in adults. Itching is most annoying when the erythema or bulla first appears, and is less distressing when they have fully developed, and in the intermissions between successive outbursts of eruption.

Occasionally the appearances assumed resemble those described by Bazin as his hydroa vacciniforme, as in the following case :—

M. D., 3 years, came on 7th September 1886. He was a healthy child, whose parents live in good circumstances in a country town. He has been troubled with what his mother calls heat spots ever since he began teething, but it is only since he was permitted to wade in a river one hot day two months past, that the eruption acquired exactly its present characters. These are, first, a minute, pin's-head spot appeared, a papule with a red areola. The areola extended, and the papule became a vesicle, flat and chambered, in fact a pock. This ruptured, a crust formed in the centre, while the flaccid walls of the vesicle remained as a collar within the still persistent areola. As the crust dried, radiating lines could be traced from it, arranged in a stellate fashion like the spokes of a wheel, and thus the resemblance to vaccinia was very close. These were found on the arms, trunk, and nates, where they were pretty numerous, and also on the legs. Itching was troublesome,

especially at night. He was ordered a bran bath at night, followed by the application of zinc ichthyol glycerine jelly, and Fellow's syrup internally in suitable doses. Three weeks later many of the vesicles had healed, few new ones had come out, distinct white marks had been left by some. In course of a short time he quite recovered, and has been free from any recurrence since.

A third form of dermatitis herpetiformis, which essentially differs in some important particulars from those previously related, is that which attacks the exposed parts of the body almost exclusively, the localities affected being nearly identical with those involved in xeroderma pigmentosum. Only a few instances have so far been published: one by Mr Hutchinson,[1] two by Unna, with notice of three more,[2] one by Dr Handford,[3] and two by myself.[4] The cases present some remarkable features in common. All have occurred in males. In Unna's cases, where three brothers and one half brother were affected, the sisters escaped. It commences as a rule in early childhood, and recurs throughout youth, lessening in adolescence, and apparently ceasing as manhood is attained. In some instances there seem to have been attacks in winter, but in most the recurrences took place from February or March to October, being suspended till the return of spring. Exposure to the rays of the sun promptly evoked an attack, and the acme of the annual seizure was always in the hot season. The eruption was erythemato-bullous, or erythemato-vesicular, and was preceded by sensations of burning, tension, or pain, rather than of itching. It implicated the ears in particular, the face, neck, and backs of the hands. Each attack was more or less sharp, was accompanied by constitutional disturbance, as well as local uneasiness, and was followed either by entire or partial relief for a time. The eruption led to scarring, occasionally to pigmentation. Hence this form also approximates to Bazin's hydroa vacciniforme.

The following case, one of those of which an account appeared in *The Lancet*, has been seen by me at intervals during nearly six years, and is in all respects a typical one:—

R. F., 18 years, a strong, well-grown lad, was brought up in the country, though for some years has been resident in town. The complaint began, according to his mother's statement, when three years old. His parents are healthy, and neither they nor any of their other children or known relations are affected similarly. The attacks came on in February, and he was liable to them till October, then he remained free till the next spring. Before an attack he felt chilly, or at least had to stay near the fire, then the

[1] *Trans. Clin. Soc. of London*, 1889.
[2] *Monatshefte f. practische Dermatologie*, 1st August 1889.
[3] *Illustrated Med. News*, 1889.
[4] *The Lancet*, 18th August 1888.

face and sometimes the hands swelled, became red and burned, but were not itchy, nor could scarcely be said to be painful. Red blotches now appeared, on which flat vesicles formed, each from a pea to a sixpence in size, the erythema persisting as an areola. The centre of the blister soon assumed a dark, reddish-brown tint, and became dry, while the margins continued vesicular like a collar. Some vesicles ruptured, and the contents dried into crusts, which were thick, friable, and yellowish, greenish, or black from blood. From their first appearance till the skin was again whole, a period of three weeks usually elapsed. Then an interval of various duration might intervene, or a fresh crop would at once appear. The ears were most seriously disfigured, some portions of the auricle having been entirely lost, imparting a crenated aspect to the edge. All the sides of the face, the neck, and to some extent the backs of the hands, are now studded with cicatrices, left behind by the blisters, and there are stains of yellowish brown pigment the size of a threepenny piece scattered over the cheeks and forehead. There are also some scars on the legs, as the vesicles formed there when he wore short trousers as a boy. The attacks are now much slighter. Thus in May 1890 only three or four vesicles appeared. He had at that time a pretty sharp diphtheria, for which he was admitted into the City Hospital. From this he made a good recovery, and several weeks later no fresh blisters had shown themselves.

In another similar case in a country lad of healthy family, and himself ruddy and well grown, the first attack did not manifest itself till he was 13, but recurred within the same limits as to time of year as in the previous instance. The scarring was, however, much less noticeable, though it was also most marked on the ears. When he had reached 19 the outbreaks were evidently diminishing in severity. Possibly the comparative mildness was owing to the disease having commenced later in life. A case occurred in Dr Muirhead's practice, and was related to me by him, where a boy suffered from precisely the same lesions, but where an important observation was made as to the influence of diet. When he was kept on vegetable food and meat withheld, the outbreaks ceased, or at least were slight: so soon as meat was given an outburst followed, again to be checked by its withdrawal. These observations may have some bearing on the leucomaine theory, to be noticed shortly, and would at all events seem to point to a disturbance of secondary digestion.

Mr Hutchinson who, like myself, has remarked the close relationship which these cases bear to xeroderma pigmentosum, or "Kaposi's Disease," [1] considers that another group of skin diseases, to which he formerly attached the name of "Summer prurigo," ought to be placed side by side with them. In these latter the eruption is sometimes erythematous, sometimes papular or abortively pustular;

[1] *Lectures on Rare Diseases of the Skin*, p. 126.

it commences in childhood or early youth ; it affects the face, neck, backs of the hands and arms particularly, but it may become nearly universal. In all cases, however, the palms and soles and the flexures of the joints remain free. It is associated with much itching, occurs almost exclusively during the warmer months of the year, and leaves small scars. These features unite even more closely this with the previously described forms of dermatitis herpetiformis, and thus we have here representatives of the erythematous, papular, vesicular, and perhaps pustular forms. "The influence of the sun in producing the eruption appeared," Mr Hutchinson says, "to be equally well marked in both cases, but the form assumed by the eruption was very different." [1]

When we consider the causation of this disease, one point is the frequency with which it occurs. I find that out of 770 cases of skin disease seen in the Royal Infirmary since the 1st of January of this year, there are six examples of dermatitis herpetiformis; two of these were in men, aged respectively 50 and 35, and four in children, the youngest being a girl aged 2 years, the other three were boys, aged between 7 and 8. In my experience it is by far most common in the male sex, only one or two women affected with it having come under my notice. It is not so infrequent in children of the female sex, but the numbers are yet too small to be worth much for statistical purposes. As to the pathology of the complaint, we may reject for the present any idea of its being due to an animal parasite ; there is no proof whatever of its being communicable. Two opinions are held : one, that it is a tropho-neurosis; the other, that it is due to an auto-intoxication by leucomaines,—those alkaloids produced, according to Gautier, in the normal metabolism of the albuminous tissues of animals independently of any bacterial agency. The two theories have been well stated by M. Hallopeau.[2] In favour of the tropho-neurosis, there are the paræsthesiæ, the symmetry of the lesions, and the recognised possibility of the evolution of vesicles in association with nervous affections, but all these characters can be found apart from neurotic disturbances. There is a striking analogy between dermatitis herpetiformis and some iodic eruptions, and it may be that from accidental or diathetic influences, the leucomaines constantly formed in the system can occasion an auto-intoxication. In support of this analogy it has been shown by Brocq, that the eruption in dermatitis herpetiformis is intensified when iodide of potassium is administered,—a fact of which corroborative evidence is afforded in two of the cases which I have related. The curious experience communicated to me by Dr Muirhead, in which the eruption came and went as meat was given or withheld, also bears strongly on the leucomaine theory. Something the same view is held by Dr Sherwell, that it is a re-

[1] *Trans. Clin. Soc. of London*, 1889.

[2] *Annales de dermatologie et de syphiligraphie*, Juin 1889, p. 564.

flex on the skin, started by septicæmia, pyæmia, or effete products from any source.[1]

The diagnosis must be based on a due estimation of all the characters of the disease, as already stated. Probably the complaint which would cause most difficulty is that known as erythema exudativum multiforme. It may be distinguished from this by the question of degree, and by positive differences in symptoms. Dermatitis herpetiformis has a much greater tendency to form vesicles, bullæ, and in rarer instances pustules; but apart from this there is the intense itching, the mode of evolution, the peculiar arrangement of the lesions, and the prolonged duration, points in which it contrasts strongly with erythema multiforme. The same features of polymorphism, of itchiness combined with the preservation of good general health, serve to separate it from pemphigus. The domain occupied at a comparatively recent time by pemphigus has notably shrunk in extent. A pemphigus pruriginosus has been described, but how far such exists distinct from dermatitis herpetiformis future observations must decide. In the course of urticaria, however, we do meet with bullæ as a further development of the wheal; but this is an accidental development: the cardinal symptoms of nettlerash are the more prominent, while in most cases local irritation will suffice to evoke typical wheals. Some medicinal rashes may simulate dermatitis herpetiformis. M. Brocq relates an instance in which salicylate of soda caused an eruption pretty nearly resembling it, but which declared its true nature by disappearing when the drug was discontinued, although iodide of potassium had been given, which would have aggravated the exanthem had it been due to dermatitis herpetiformis.

The treatment cannot be said to be yet satisfactory. In some cases baths of potassa sulphurata, two ounces in thirty gallons, with the addition of two or three pints of freshly made starch, and taken for a quarter of an hour at a temperature of from 95° to 98° at night, have certainly afforded much comfort, but in other cases, particularly if the heat named has been exceeded, have seemed to intensify the itching. In one instance this was manifestly due to the water being too hot, so that it stimulated in place of soothing the skin. Two applications are of value after the bath. One is the calamine lotion, to six ounces of which a drachm of boracic acid and one of carbolic acid, or of the liquor carbonis detergens, have been added. Or the following lotion, which both soothes and cools, is advantageous:—R. Liq. calcis, Ol sesami, ana uncias tres.; Cretæ præp., Zinci oxidi, ana semi unciam; Acidi salicylici, drachmam unam. In several cases, more particularly in children, sulphur ointment, which may even be made as strong as three drachms in the ounce, does more good than anything.[2]

[1] *Journ. of Cutan. and Genito-Urinary Dis.*, Dec. 1889, p. 458.
[2] Duhring, *International Journ. of Med. Sciences*, June 1890.

Internally arsenic best restrains the evolution of the vesicles and bullæ, and the greatest benefit has been obtained by me from the pills of arsenious acid, one-fiftieth to one twenty-fifth of a grain thrice daily. Arsenic has been found to check the toxic effects of iodide of potassium, and is to some extent its antidote. Arsenic has the effect of increasing metabolism, but at the same time presents the curious anomaly, that though it apparently restrains the formation of vesicles and bullæ in dermatitis herpetiformis, it occasionally seems to provoke another vesicular eruption, viz., herpes zoster. The direct connexion of this latter with special nerve disorders is well known and accepted, so that this action of arsenic in dermatitis herpetiformis also favours the leucomaine theory of origin. We may by such measures bring the attack to a termination, but we can by no method yet discovered protect from a relapse or a recurrence.

Schwimmer of Buda-Pesth records a case[1] in which thiol (a synthetically prepared substitute for ichthyol) was employed externally with success. Other remedies had been used without effect for three months; the patient was now painted regularly twice daily with a one in three solution in water for two or three days, and the skin then carefully washed with pure water. It was found that the vesicles and bullæ had disappeared even in this short space of time, being replaced by scurfs of thiol, while the skin below showed only moderate pigmentation. Since this was published no instance of dermatitis herpetiformis has come under my notice, so that I have been unable to test the influence of thiol for myself.

Prof. Annandale wished to thank Dr Jamieson for his interesting paper. He did not profess to any special knowledge of skin diseases, but he expressed the hope that, although the disease so clearly described by Dr Jamieson was, at present, not very amenable to treatment, it might soon become so.

Meeting II.—December 3, 1890.

Professor A. R. Simpson, *President, in the Chair.*

I. Election of New Members.

The following gentlemen were elected Ordinary Members of the Society:—Robert Thin, M.A., M.B., C.M.Ed., 3 Argyle Place; George M. Robertson, M.B. Ed., Senior Assistant Physician, Royal Asylum,

[1] *British Journ. of Dermatology*, Sept. 1890; abstract from *Wiener klin Wochensch.*, No. 18, 1890.

Edinburgh; J. M. Balfour, M.B. Ed., 18 Walker Street; James Hutcheson, M.D., F.R.C.S. Ed., 8 Nelson Street; Dr A. Cowan Guthrie, 53 Charlotte Street, Leith.

II. Exhibition of Patient.

Mr A. G. Miller exhibited the following case of EPILEPSY, from injury to the head, cured by trephining:—A. S., married, æt. 42, wire-weaver; chief amusement, fishing from end of Leith Pier. Injury to head, November 1884. In dark, stooping to take up his tackle, was struck on top of head by heavy sinker (1 lb.); was stunned, but not unconscious; noise like a shot heard. Scalp wound dressed at Leith Hospital, healed by first intention. Cicatrix tender. No headaches. Only previous injury was a stab on right side of neck over middle of sterno-mastoid. First fit in middle of night, 26th June 1888, more than three and a half years after injury. Second fit next day. Speech affected after this. Right side most affected in fits. Character of fits when admitted to Edinburgh Royal Infirmary (Ward 31, under care of Dr Wyllie), July 1888: Conjugate deviation of eyes to right and upwards, right side of face convulsed, head turned to right, general convulsive movements; no unconsciousness, but aphasia complete. There were about forty to fifty fits daily. Operation, 10th July 1888. Trephining. Incision made over cicatrix, on the left side of the top of head in front. Three circles removed on left side in a line from area 12 to area 6 (Ferrier). Nothing special was found to account for fits, though a needle was passed freely through the brain substance. After operation, fits much the same in frequency, but less severe; aphasia less marked. After three weeks fits diminished, and after six weeks disappeared, as also the aphasia. Patient went home September 5th, and was able to resume work a month later. Patient has remained well and able for his usual work ever since, having had no fits except once, three months after leaving Hospital, when he had a fit accompanied with aphasia, brought on apparently by grief at the death of a child.

III. Original Communications.

1. SOME REMARKS ON THE NEW METHOD OF TREATING ENLARGED PROSTATE BY DR BOTTINI, Professor of Operative Surgery in the Royal University of Pavia.

By Dr Morotti.

I MUST thank the President and the members of the Medico-Chirurgical Society of Edinburgh for the great honour they have done me in giving me this opportunity to speak about a discovery of my Professor of Surgery, asking them at the same time to

forgive a stranger for his language, and to look only to the great attraction of the subject.

Hypertrophy, or rather the enlargement of the prostate, is of such common occurrence as to be called the heritage of old age. Every enlargement of the prostate does not prevent the free emission of urine; the total and eccentric usually do not produce disturbances in urination, while, on the contrary, the partial and concentric produce strictures, more or less serious, according to the position and size of the enlarged lobe.

The causes which usually induce the prostatic strictures are of the same kind as those which induce the organic strictures. The advantages of the palliative cure are more imaginary than real. Periodic and permanent syringing is accompanied by such serious consequences that it can hardly be said whether the remedy is not worse than the disease. From the mere rubbing in with ointments, to the complete extirpation of the gland, we have a series of progressive plans, from the simplest to the most daring.

To effect a cure of ischury from prostatic strictures one must understand and meet the diseased condition which produces the enlargement. In prostatic strictures the ischury is occasioned by a mechanical obstacle to the emission of the urine. Now, the therapeutic direction must follow two indications,—*Firstly*, To increase the expulsive power, so as to overcome the obstacle; *secondly*, to remove, or at least to lessen the obstruction, so as to restore the regular passage of the urine.

The surgeons of all times have endeavoured to carry out the second rather than the first of these two questions, and have thought of many different ways. The apparatus invented by Dr Bottini completely carries out the idea of making an artificial passage for the urine in the body of the same prostate. The passage of the urine being restored, hypertrophy of the prostate does not any longer entail danger to life, therefore it is useless to take away an organ which, although abnormally enlarged, yet does not affect the general work of the entire body. Therefore I am anxious you should be persuaded that the therapeutic idea is not that of melting or otherwise destroying the prostate, but that of making a new passage for the urine to replace the passage which has been obstructed by the abnormal enlargement of the gland. The way of fusion and incision of the prostate by thermo-cautery in such conditions seems rational, both as to its immediate effects and as to its ultimate consequences. Therefore we not only need to divide the prostatic growth, as the prostatic incisor of Mercier does, but we require to keep it open, making at the same time an aperture inside, which will not be quickly closed up, but leaves a permanent groove through which the urine can flow.

The instrument of Dr Bottini resolves the problem of the practical application of such an idea. The problem was how to heat a certain portion of the instrument, so as to bring it in front of the

diseased enlargement which is to be treated, limiting the caustic effect to burn a particular spot, without injuring the urethra and the bladder.

After long and diligent experiments on dead bodies and on living animals, Dr Bottini succeeded in making the instrument which I now have the honour of showing to this honourable assembly, and which answers thoroughly the object proposed. The "cauterizatore termo-galvanico" is composed of a metallic tube resembling the catheter of Mercier, internally divided into four compartments for the passage of the conducting electric wires, and the tube for the water, which, entering into one of the canals, runs to its extremity, then passes to the other canal, and flows out at the free end, keeping the whole instrument at a constant low temperature. The conducting wires end in two little plates, one-third of a centimetre broad and two centimetres long, placed parallel to each other at the distance of one millimetre, and joined at the bow towards the beak of the instrument, from the curve of which they are separated one centimetre. These plates must get heated to a dull red—*the only colour*—which insures destruction of the tissues without causing a single drop of blood. The flow of cold water, keeping all that part of the instrument constantly cold, so that it may not have a caustic action, prevents the epithelium and the mucous membrane of the bladder being injured, while cauterization of the part of the prostate is being effected.

Here I must ask you to allow me to offer to your consideration some rules for the operation. It is always better to accustom the patient to the use of the instrument before the cauterization, so that its introduction may be easily and quickly done, and the enlargement may be reached at once by applying the cautery exactly to the designed spot. The rule of accustoming the urethra to the passage of the "cauterizatore," so that the instrument may slip by its own weight into the bladder, as Civiale happily expresses it, "C'est l'urethre qui doit avaler la sonde," and so that we can bring the heated portion against the part to be destroyed, is of vital importance. As the patient has been placed in the position used for lithotripsy, unless the bladder be completely empty, we don't need to inject water. The operation is not painful; Dr Bottini uses chloroform only occasionally; nevertheless we must insure that the patient will be kept still during the operation, otherwise by a sudden movement the instrument might get displaced, and come against parts which ought not to be touched.

The operative technique consists of three parts:—

Firstly, We introduce the instrument, and place the cautery against the part of the enlarged prostate to be burned.

Secondly, We get the cautery heated, and we cauterize to a sufficient degree.

Thirdly, We withdraw the instrument.

In regard to the first, we introduce the instrument into the bladder according to the classical rules prescribed in urethral catheterism. Then we must turn down the beak, as we do in the methodic exploration of the fundus of the bladder, and pulling the shaft so as to hook the neck, we may carry the cautery against the proper spot. If any doubt arises, the position of the instrument may be ascertained by a finger introduced into the rectum. The instrument being properly placed we allow the water to come in, which can be done by suspending a common pail, or an irrigator of Esmarch, at a certain height, capable of holding six or eight litres of water, at a temperature of about 40° to 50° Fahrenheit.

The battery which Dr Bottini uses is a Bunsen's, with two liquids in the improved manner of Pischel, and lately again improved by Dr Bottini, by the introduction of the accumulators, as he has fully described in his article in the *Lancet* of the 20th September, which I would request my audience kindly to read, so as to complete the subject.

The heating of the cautery must not exceed the dull red; if it does exceed this, and becomes white, the tissues will be too quickly destroyed, and most probably hæmorrhage will ensue.

With repeated experiments on dead bodies and on living animals, Dr Bottini obtained, in one minute, by the cautery being heated to the red colour, an eschar about the thickness of a centimetre, and the action being prolonged for another minute, the thickness has become doubled, when he could raise the temperature by one degree, changing the commutator of the battery.

To insure the eschar being formed as indicated, the heated portion of the instrument must be placed against the part to be burned, raising the shaft and bending it gradually, so that the resistance of the diseased tissue having been overcome the beak can be embedded in it. We must then stop the farther production of heat by shutting off the current, and let it remain for two or three minutes, so as to give time for the plates to cool, so as to render harmless the withdrawal of the instrument, which will be done in an opposite sense in the same manner as when it is introduced. We do this in order to prevent the risk of the eschar being brought away with the withdrawal of the instrument.

For the after-treatment we use the soft catheter of Nelaton, always provided that the patient cannot pass water by himself. Dr Bottini wrote me that a few days ago he had to operate on an old man, suffering from prostatic ischury for more than five years, who after twenty-four hours from operation was able to pass water without artificial assistance. The eschar is completely gone by the tenth, twelfth, or fifteenth day.

"Dr Bottini has operated after this method on fifty-seven cases, two of which ended fatally; these, however, were during the early days of the operation, before the instruments had been brought to their present state of perfection. In thirty-two cases a perfect

cure had been obtained, in eleven there was an improvement, and in only twelve was no benefit observed. These results in an affection which was so rebellious to ordinary treatment, form a powerful *argument in favour of the method;* and the author hoped soon to see it adopted more generally by surgeons than it had yet been."

As this new method is destined to work an entire revolution in the art of curing ischury from the prostatic enlargements, it is most important that this process be understood in every particular, and carried out with the most minute and diligent care. It is true that the recent improvements introduced by Dr Bottini in the apparatus may be said to remove many dangers due before to the use of imperfect instruments; but at the same time, I must point out that the galvanic cautery requires the greatest care, otherwise the patient will be liable to great dangers.

In conclusion, when the galvano-cautery is used in the manner described, and under favourable circumstances, success is sure to follow. The important improvements recently introduced in the apparatus renders the operation simple and takes away all danger, so that it is not reserved for specialists alone, but is open to all surgeons. There must also be a most intimate knowledge of the practical part, and certainty in carrying it out. To be justified in using it, the dysuria must be constant and lasting. If, however, it is well known that in every surgical undertaking it is necessary to have a thorough knowledge of the instruments to be used, it is even more indispensable in an apparatus so delicate in its construction, and of such powerful caustic action.

The apparatus has been highly approved, not only by Italian surgeons, but by foreigners. Bruce Clarke of London uses it, and praises it greatly. Allow me to say that this instrument marks a new and decided advance in the art of curing this distressing and intractable class of diseases.

I hope, therefore, that you, being satisfied of the importance of the subject and of the efficacy of the instrument, will make a trial of it, and salute with me the inventor, who has done such a good thing for science and for suffering humanity.

2. PERSONAL IMPRESSIONS OF KOCH'S TREATMENT AT BERLIN, WITH EARLY NOTES OF CASES TREATED IN THE ROYAL INFIRMARY OF EDINBURGH.

By R. W. Philip, M.A., M.D., F.R.C.P. Ed., Physician to the Victoria Consumption Dispensary, and Assistant Physician, Royal Infirmary, Edinburgh.

I had proposed to-night making a communication regarding an extended research into the local therapeutics of the respiratory apparatus. But the publication by Dr Koch on the 14th November of "A Further Communication on a Remedy for Tuberculosis,"

and the opportunities afforded by a visit of ten days to Berlin for studying the results obtained, have led me to delay the other in favour of a summary of the impressions produced by this visit. If these should appear imperfect or disjointed, I beg you will allow the short time which has elapsed, and the press of work during the few days since my return, to be the excuse.

I have had an opportunity of observing, more or less fully, some 200 cases in different hospitals, and what I have to say is based entirely upon my personal notes. I take this opportunity of testifying to the courtesy and kindness displayed by the physicians and surgeons in charge.

The fluid—of which, through the personal kindness of Dr Koch, I have been able to bring home a small quantity, and of which I show you a few drops—is transparent, and of brownish colour, resembling an East India sherry. Of its chemical constitution little can be said definitely. But from statements made by Dr Koch to his Excellency Herr von Gossler, the Minister of Education, and other incidental references, it is probable that it contains one or more of the chemical resultants from the growth in given media of the tubercle bacillus. From what I learned at the Hygienic Institute, it is clear that the process of elaboration and separation is difficult. Koch has stated that it would take months for a good worker to become thoroughly familiar with the method of preparation. This, along with the necessity for uniformity of strength, explains the apparent delay in the distribution of the fluid. There seems every likelihood that, meanwhile, the German Government will keep the preparation and distribution in its own hands.

In its undiluted state, the fluid may be kept indefinitely without change. But when diluted for injection—as it is, usually, with distilled water—it is liable to decomposition, the fluid becoming turbid. This difficulty may be overcome by diluting with a half per cent. solution of carbolic acid. Boiling causes no precipitation, but the addition of alcohol does.

For injection it is generally used in solutions of 1 to 10, or 1 to 100, or 1 to 1000. The proportion of the solution is, of course, of less importance than the actual dosage. For most internal conditions, such as phthisis, empyema, etc., the treatment is commenced with 0·001 gramme (in delicate patients and children, it may be, with 0·0005 gramme). Comparatively early a relative tolerance is established, and the dosage is increased gradually up to 0·01 gramme or more. For more superficial conditions, such as lupus, tubercular adenitis, articular tuberculosis, etc., the dose begins usually at 0·01 gramme for the adult, and this is gradually increased up to, say, 0·1 gramme. The rate of increase I found to vary considerably in the hands of different operators and with different patients.

The fluid is introduced subcutaneously. It has apparently no action by way of the stomach. Fräntzel and others have tried its

introduction by way of the respiratory passages; but while it was found to be operative, greater difficulty was experienced in regulating the dosage. The seat of injection is quite independent of the seat of lesion. That selected by Koch (at first for convenience, and that the patient might not know what was going on), in the loose fold between the shoulder blades or in the lumbar region, is usually adhered to, though I have frequently seen the injection made into the leg and arm, apparently with equally good result. The instrument in general use is Koch's bacteriological syringe. It has been sufficiently described in the medical papers, so that I need not enter into details regarding it. For rapid clinical work it has disadvantages, which can, in my judgment, be avoided by the use of one of the recently perfected hypodermic syringes, without any risk of harm to the patient, if cleanliness be properly attended to. Already in some of the clinics other syringes are in use. Meanwhile, in the Royal Infirmary, I have preferred to continue Koch's syringe, and rigidly to follow all his instructions till the method be fully tested. The injection is practically painless. I have never seen local disturbance occur at the seat of injection, saving slight pain when the patient happens to be percussed over the point of entrance of the needle, it may be on the succeeding day.

Effects of Injection.—On a healthy subject, that is, a non-tubercular subject, injection of so comparatively large a dose as 0·01 gramme produces practically no result. There may be passing, hardly appreciable, *malaise*, with, perhaps, rise of temperature to 100° F. To produce serious symptoms on such a subject, so large a dose as 0·25 gramme is required, as tested by Koch in his own person, when three to four hours after injection there supervened pain in the limbs, fatigue, inclination to cough, and difficulty in breathing, which speedily increased. In the fifth hour a prolonged rigor ensued, and, about the same time, sickness, vomiting, and rise of temperature to 103°·3 F. After twelve hours these symptoms abated. The temperature fell till next day, when it was normal. A feeling of fatigue and pain in the limbs continued for a few days, and the site of injection remained similarly slightly painful and red.

In most of the cases of lupus which I saw treated the dose was commenced with 0·01 gramme, except in case of children, or when, in addition to the lupus, there was evidence of other tubercular processes. If internal processes, pulmonary or otherwise, complicated the case, the dose was that given for internal cases. With such a dose, 0·01 gramme, in practically every case results of a most striking character ensued, conveniently divided into general and local.

General Reaction.—This begins to be manifest from four to six hours after injection. It consists in the onset of fever—the temperature rising, often rapidly, to from 102° F. to 105° F., or even higher. Associated with this there is an increase in the pulse

rate and in that of the respiration, the latter, as a rule, relatively greater than the former. In one case, where, however, there was undoubted lung complication, and the patient had a correspondingly smaller dose, the respiration became 60 per minute at the height of the reaction. (I have also seen this rate attained in a case under treatment in the Royal Infirmary of Edinburgh.) The patient lay panting for breath, but was not so livid or otherwise disturbed as when a corresponding rate occurs, say, in croupous pneumonia. The pulse, while rapid, was fairly full and perfectly regular. Associated with the increase in respiration, and sometimes apart from it, there is coughing, often troublesome, accompanied, in many instances, by expectoration, even when there has been none previously. Sickness, vomiting, and general *malaise*, of varying degree, occur. In some instances diarrhœa was noted. In some dozen cases I saw a distinct exanthem—something between a scarlatina, a roseola syphilitica and measles, but in others more papular. (I have seen this occur twice in the cases I have since injected at Edinburgh.) In a fair proportion of cases an icteric tinge of the skin was distinctly evident. A few appeared to me unusually pale. In a few instances epistaxis was noted.

More serious manifestations consist in loss of consciousness and delirium (which I have seen at least a dozen times in young children at Berlin, and once in Edinburgh), followed by a condition of somnolence, lasting many hours it may be. In some instances, the somnolence occurred apart from the other cerebral condition.

As a rule, the general reaction begins to decline at the end of twelve hours or so; sometimes, in one or other manifestation, it continues for twenty-four hours or longer. The complete cessation of general symptoms affords a guide to the desirability of repeating the injection. Often a day or two, it may be longer, is allowed to elapse before the second injection. The remarkable feature, *quâ* the general reaction, is that a repetition of the same dosage produces generally distinctly less serious results.

The *local reaction* is best studied in lupus. It becomes manifest, with practically absolute certainty, in the affected area or areas, wherever situated. It appears at a varying period from six to twelve hours after injection, and consists in swelling—the nose, for example, becoming two or three times its normal size—and redness of the diseased part. A zone of redness of varying breadth also surrounds the lupus area, and, in well-defined cases, a whitish ring immediately between the red zone is distinctly traceable. This is often preceded—it may be some hours—or accompanied by a subjective feeling of tension and burning. A slight exudation soon begins to ooze from the surface, the yellow drops of which run together, dry, and form crusts. This incrustation grows in thickness, until it tumbles off in, perhaps, a week or ten days (in one case in the Royal Infirmary part of the scab began to break up shortly after the second injection). Sometimes pus

tends to collect below, and it is convenient to loosen the crust to afford free vent. This may easily be accomplished by the use of oil or other softening agent. In many cases where it was allowed to fall off naturally, I have seen it disclose a smooth, delicate surface of pale red colour. In some cases where I saw it picked off, the sore presented the appearance of a granulating surface.

I have seen some six or eight cases of lupus to all intents and purposes cured after from six to ten injections. The frequency with which the injections are repeated I have found to vary with different operators. Koch, in his original statement, speaks of allowing the reaction after the first dose to come to an end entirely, and, after a week or two, again injecting 0·01 gramme; but at some of the hospitals the dose was repeated more frequently, the time being fixed by the general state of the patient, and, particularly, the disappearance of all signs of general reaction.

In connexion with lupus, it is most interesting to note that the local effect is not confined to the place *evidently* affected. I have seen this frequently illustrated. I have seen swelling and redness appear at points—perhaps in the extremities—where no lupus had been observed or suspected. After one or two injections patches of lupus have made their appearance, as if the tubercle nodules were lying latent, too deeply buried to be noticeable, till accentuated and brought to the surface, as it were, by the specific. Further, several patients with lupus have, after injection, complained distinctly of pain in one or other joint, or a gland has become definitely swollen, suggesting that a focus of tubercular disease was present where it had not been possible to diagnose. Further, in one or two cases, local changes in the chest, subsequent to treatment, suggested that pulmonary tuberculosis, hitherto undetected, was present—a fact to which, perhaps, the supervening sputum pointed. It must, however, be borne in mind that sometimes this last symptom developed after injection in apparently healthy persons.

Before leaving lupus, I should add that I have seen several cases of lupus affecting the mucous surfaces of the palate, pharynx, and tongue react in a similar way, and show a similar tendency towards cure.

Glandular conditions are treated in the same way; the general reaction need not be described again (see above). The local reaction manifests itself frequently by increased swelling, which continues for twelve to twenty-four hours or longer, with perhaps an accession of tenderness. The swelling then tends to diminish till the glands become smaller than before; but I have seen no case where the glands were so reduced in size as to indicate the hope that they would thus be cured without the intervention of the surgeon to remove the presumably necrotic tissue. In a case treated, since my return, in the Royal Sick Children's Hospital here, the primary increase was well marked, and the subsequent decrease

was very striking. In this case there was much free exudation from the affected areas through the numerous sinuses which existed; these, prior to injection, had scarcely been discharging at all.

In articular conditions the process is necessarily much slower, and in the comparatively short time available for observation it has been impossible to follow those far. In a large proportion of cases, in addition to the general reaction, a definite local reaction was traceable, manifested, it might be, by increased swelling or tenderness—sometimes both—sometimes by abnormal fixation, to be followed, in the three or four cases which appeared to me to be making progress, by loss of tenderness, by increased power of voluntary movement, and less discomfort when free passive movement was attempted. Dr Levy showed one or two cases of tubercular arthritis in which he noted evident improvement. In Professor von Bergmann's clinic, during the ten days I visited the wards, I could recognise definite improvement in those directions in one or two cases. As a result of injections of several cases of glandular and articular tuberculosis other situations were sometimes indicated as probably seats of tubercular action, from the appearance of more or less characteristic local reactions.

With regard to internal conditions, I have had an opportunity of studying a very large number of cases of phthisis pulmonalis, phthisis laryngea, pleurisy, empyema, tubercular diarrhœa, etc.

In most instances the cases are being observed with great care. The state of the patient is carefully noted for a day or two before the inoculation, and a thorough examination of his system made, so as to include as much as possible every tubercular manifestation. The sputum is examined with care and frequently reported on. The temperature, pulse, and respiration are noted for twenty-four hours prior to injection.

Special clerks, or other trained assistants, are told off to make note of every change, and the pulse, temperature, and respiration are noted every hour, or once in three hours (in some cases). Very much the same method is being followed in the Edinburgh Royal Infirmary, where the resident medical officers have been most assiduous.

In Professor Gerhardt's wards (where, through the kindness of the physicians, I had excellent opportunities for observation) the presence of the tubercle bacillus in the sputum was taken as the test of the case being presumably one of tubercular phthisis. Cases were included for observation only where the condition was reasonably early, and where, in other words, it might fairly be supposed that the tubercle bacillus was not abetted in its work of destruction by other organisms, which have been described in connexion with late processes.

Speaking generally for these internal conditions—and particularly for phthisis—the treatment is commenced with 0·001

gramme in the adult (or, in some strong, healthy subjects, 0·002 gramme).

In almost every case where there was reasonable ground for diagnosing phthisis the characteristic general reaction occurred. This differed in no respect from that witnessed in connexion with the local processes, except that the severer symptoms, such as rigors, were generally absent when the dosage was kept small. The changes in pulse, temperature, and respiration have been already noted. Headache, lassitude, and occasionally vomiting occurred in many instances.

The local reaction, which occurs in from six to twelve hours, manifests itself by increased respiration (often more marked than in the ordinary general reaction), pointing to change in the lung, by a subjective feeling of dyspnœa, often by a peculiar sense of oppression and tension, as if the lungs were too big (as one patient expressed it), by cough, or increase of cough if already present, by increase of expectoration, which usually became more frothy and fluid (in some cases more uniformly purulent). In six cases hæmoptysis followed, but only in one case to any important extent—and in a patient liable to it. But more interesting still is the change which occurs to physical examination. In a fair proportion of cases the percussion area of dulness was found to be increased, and in a considerable number the auscultatory phenomena of crepitations became more numerous. In some five or six cases I examined, where practically no dulness could be determined prior to inoculation, it became evident after, as did also crepitations not previously audible.

With regard to final results in these cases, especially in phthisis, it is premature to say much. In very advanced cases, of which I had the opportunity of studying about a dozen, I cannot say that I was satisfied of any definite improvement. The *post-mortem* results of the two or three fatal cases, so far as they have been communicated, point, however, to a local influence having been exerted on the affected organs.

In less advanced cases, but still cases where, prior to the special treatment, physical examination revealed considerable areas of percussion and abundant crepitations, and when the patient suffered from loss of flesh, hæmoptysis, night sweats, and anorexia, the report must be more hopeful.

In a considerable proportion of these I think we may safely admit that very definite amelioration can be affected. I observed a number of cases where night sweats had disappeared, where the cough was practically gone, where there was a diminution or cessation of expectoration, and where the patient's subjective condition was better, and a few where there was a gain of several pounds in weight. In very few cases was I able to trace important improvement in the physical signs; but the time was too short for this, and no case has, so far as I was able to learn, been re-

leased from treatment as cured. Still I think, pending further observation, we are bound to fall back on the authority of Koch, who states categorically that "within four to six weeks patients under treatment in the first stage of phthisis were all freed from every symptom of disease, and might be pronounced cured."

With regard to the change in the tubercle bacilli, very varying statements are made by different observers. Professor Fräntzel states that in a considerable number of cases the bacilli disappeared altogether, and that even when they did not they became smaller and thinner, as if they were starved; sometimes they became altered in form, becoming biscuit-like; sometimes they were broken up or apparently partially disintegrated.

To summarize a little, then, there can be no question that the fluid, whatever its nature, has a most defined *specific* action on the tubercular process, wherever situated. It seems to have the power, when introduced into the circulation, of finding its way straight to the seat of tubercular disease, however obscure or ill-defined this may be to external examination. Many of the cases I have already cited illustrate this well—the appearance of fresh lupus patches, the indications of pain or swelling of joints which had not previously been detected, and the reddening of old tubercular scars. Most striking in this connexion are some dozen cases where I had the opportunity of examining the larynx after one or two injections. Here, as I was informed by the physicians of the wards, no change had been detected prior to inoculation (most of the cases were phthisis laryngea). Subsequent to inoculation there certainly was a distinct indication of disease. In two or three, this was shown by swelling of the aryteno-epiglottidian folds; in three cases, by excoriation, and even ulceration of the laryngeal surfaces, and, in one instance, by ulceration of one of the upper rings of the trachea.

Again, in three cases (lupus, supposed pernicious anæmia, and a doubtful apical catarrh) physical signs were detected after injection, and an abundant expectoration, not present before, was established.

In two cases, ultimately determined to be cancerous—one of the larynx and one of the face—the final conclusion was reached in virtue of the definite absence of reaction. This *diagnostic* value is also illustrated by some three cases of pleurisy, two with effusion, where injection produced a most manifest reaction, proving the presence of a tubercular process somewhere, and presumably affording corroboration to the view that a large proportion of these pleural processes are tubercular. In one case under treatment in the Royal Infirmary, no definite reaction, general or local, has occurred after four injections, although the dose has been increased to 0·01 gramme.

To what I have said of its *curative* value I do not propose to add anything. This has been sufficiently illustrated in the less severe and slighter forms. As to its limits and its duration, the

time is yet too short to enable us to speak. But it is only fair to admit that up to a certain point this has been established.

The facts which have been made public regarding the effects of treatment in late cases of phthisis are discouraging, undoubtedly, but they form the strongest argument, as Koch has pointed out, for far more conscientious care in the early diagnosis of such cases, and for the use of all the means which later research has suggested, such as the careful examination of the sputum for the tubercle bacillus.

The cases which, through the kindness of a number of the physicians and surgeons of the Royal Infirmary and Royal Hospital for Sick Children, I have been enabled to place under treatment, and to which reference has here and there been made above, include eight cases of phthisis, three of pleurisy, two of lupus, two of tubercular adenitis, one of tubercular affection of the knee-joint, one of cardiac disease with obscure lung complication, and one of probable genito-urinary tuberculosis. Without entering into further details, it may be well to say, that all we have seen corroborates the statements made in Koch's original communication.

The President thought Dr Philip was to be congratulated on the opportunity that he had enjoyed of observing the cases that had been put under treatment by Koch's method, and also that Professor Koch had done him the honour of entrusting him with some of his lymph for the purpose of testing its power in Edinburgh. But he thought, at the same time, that the Society was to be congratulated on having amongst its members a young physician of enterprise and ability, who had shown himself capable of making such exact and careful observation of the cases, and presenting such an interesting and lucid analysis of them as they had heard from Dr Philip. The gentlemen in charge of the patients who had been subjected to treatment in our Infirmary were present, and he (the President) hoped they would state their impressions of the new therapeutic agent.

Professor Grainger Stewart congratulated the Society and Dr Philip upon the fact that he had been able to receive from Dr Koch a supply of lymph at a time when it was scarcely possible for one to get it, and he thought that to a large extent the granting of a supply to Dr Philip was a recognition of the work that he had done in connexion with tuberculosis. With regard to the use which he (Dr Grainger Stewart) had made of the lymph thus obtained by Dr Philip, he could only in the meantime say, that the results which it was bringing out in his patients precisely corresponded to those described by Professor Koch and others. In his case of lupus the temperature had risen, eight hours after the inoculation, to 103°, while the whole lupus surface, cicatricial as well as more active, had become reddened and swollen. In his

case of phthisis, the dose of the lymph administered was very small, and no rise of temperature had resulted; still the patient had some sense of uneasiness in the chest, and numerous coarse crepitations had manifested themselves in an area of lung where no crepitation had existed prior to the inoculation. These and subsequent cases would be very carefully noted, and the results in due time communicated to the Society.

Dr Brakenridge said, that through the kindness of Dr Philip he had been able to note the results of inoculation with Koch's fluid in two cases of phthisis and one of pleurisy in his wards. In only one of these—a case of advanced phthisis with a cavity in the left apex—had any definite reaction as yet resulted. This case illustrated fully the various phenomena of the reaction, as described by Koch and observed by Dr Philip. Very notable was the increase in the rapidity of the respirations as compared with that of the pulse, the increase in area and intensity of the lung signs, and the marked alteration in the characters and quantity of the expectoration, which became much more abundant and watery. The pleurisy case was one of two months' duration, with very great effusion, and was selected for diagnostic purposes, as it had been asserted by certain French pathologists that tubercular changes are present in all cases of pleurisy. Of course it was impossible to draw any conclusions as to curative power or theorise from such initial trials; but if Koch's fluid really did possess the power to search out and produce the changes in tubercular tissue claimed for it, it might be expected that by means of it much light would be thrown on the vexed question as to whether the tubercular bacillus was primarily, or even mainly responsible for the production of all or any of the forms of phthisis. Dr Brakenridge wished to ask Dr Philip if it was not thought probable that in some of the cases in which symptoms of severe brain disturbance had followed the injections of the fluid, these symptoms might be due to irritation set up in latent tubercular disease of the meninges?

Prof. Gairdner said that he had listened with much pleasure to Dr Philip's statement, inasmuch as it afforded a strong personal corroboration of what they had all learned with such deep interest in the now universally spread communication of Prof. Koch. With regard to the latter, he (Prof. Gairdner) had himself lost no time in imparting the first impressions he had derived from it to his class. In doing so he had not, indeed, been able to add anything from his own experience, but had insisted on the character of Koch's previous career as an admirable lesson in patient and cautious, as well as brilliant research and discovery, well entitling him, in reference to the present subject, to much more than ordinary acceptance. The theory of the process was not as yet fully disclosed, so that we had chiefly to do with the results, so well and so clearly stated by Dr Philip. In the case of lupus, it might

already be said to be clearly ascertained that the direct action of Koch's fluid was not upon the bacillus tuberculosis, but upon the soil, *i.e.*, the already altered tissue cells, in which it grew and flourished. By the very remarkable power of selection which the new substance displayed, it seemed to act partly in the way of destroying, partly in the way of renewing and improving this soil, so as to make it in the end unfit for sustaining the life of the parasite, which nevertheless (as indeed Koch himself has stated) was not directly killed by the injections. In other words, the process was similar, more or less, to that which occurs in agriculture, when noxious weeds or undesirable overgrowths had to be got rid of in a field or on a hillside; by ploughing up and manuring the soil, and by diligently maintaining it in a state fit for cultivation, the weeds may be gradually got rid of, though often not till after a long time has elapsed. In dealing remedially with favus and other parasitic skin diseases the same difficulty is experienced, and in his (Prof. Gairdner's) opinion it would be an error to expect from Koch's treatment in lungs already saturated with tubercle the immediate curative results that some were looking for. Many disappointments and considerable delay must needs take place before the curative value and limits of the treatment could be said to be established in phthisis pulmonalis and internal tuberculosis generally. But the blame of this, if it occurred, would not rest with Dr Koch, who in his own personal statements had taken the greatest pains to make all the necessary reservations, at the same time showing that the new treatment was not to be employed rashly in very advanced cases, nor even in early cases, as superseding or opposing other treatment, but only in accordance with the principles underlying all successful medication in this and all other diseases. He was prepared, accordingly, so soon as he obtained the necessary lymph, to follow in the footsteps of Koch; and he should do so hopefully, and with all the love and appreciation inspired by the manifest conscientiousness, modesty, and truthfulness with which the announcement was made; and he trusted that the ultimate results might be such as fully to justify the anticipations of such observers as Dr Philip.

Dr R. W. Philip thanked the members for the cordial way in which they had received his paper. With regard to the curative value of the new method, he was grateful to Professor Gairdner for emphasizing the necessity for caution in the statement of one's anticipations or results. It had been his endeavour to couch the communication in such terms as would indicate a desire to be strictly impartial and judicial, while yet hopeful. Professor Gairdner had expressed himself as willing to rely on Dr Koch's statements. Dr Philip asked no more. But he would remind the Society that with regard to phthisis—about which there naturally was the greatest difference of opinion—Dr Koch stated in his original communication that "within four to six weeks patients

under treatment for the first stage of phthisis were all free from every symptom of disease, and might be pronounced cured. . . . . These results lead me to believe that *phthisis, in the beginning, can be cured with certainty by this remedy.*" With Dr Koch, he would again emphasize the onus which now lay more than ever on practitioners in respect of early diagnosis. As to head symptoms, Dr Brakenridge's remarks were very suggestive, though he was not aware that there was yet sufficient evidence for associating these necessarily with meningitic or other similar conditions.

3. ON A CHARACTERISTIC ORGANISM OF CANCER.

By William Russell, M.D., F.R.C.P.Ed., Lecturer on Pathology in the School of Medicine; and Pathologist to the Royal Infirmary, Edinburgh.

For some years past I have been occupied, so far as my routine duties and other researches would allow me, in tracing the mode of growth of cancer in different organs. By this study I hoped to map out the steps of the process, and, by learning the manner of its growth, perhaps to obtain an insight into the factors determining the departure of the tissues from their normal behaviour and arrangements. In the course of these studies I met with appearances which I could not fit into modes of cell growth and nuclear proliferation, and one of these cases so puzzled me that I asked my principal pathological assistant, Mr W. F. Robertson, to experiment on it with every possible combination of stains, with a view to the possible differentiation of some of these structures. His attempts were soon successful, for by a process of double staining, first with fuchsin and then with iodine green, without passing the sections through any specially decolorising agent, the iodine green replaced the fuchsine in everything, with the exception of certain bodies.

Directions for Staining.—1. Saturated solution of fuchsin in 2 per cent. carbolic acid in water. 2. One per cent. solution of iodine green (Grüber's) in 2 per cent. carbolic acid in water. Place section in water. Then stain in fuchsin ten minutes or longer. Wash for a few minutes in water. Then wash for *half a minute* in absolute alcohol. From this put the section into the solution of iodine green, and allow it to remain well spread out for *five minutes.* From this, rapidly dehydrate in absolute alcohol, pass through oil of cloves, and mount in balsam.

The fact that I had observed special structures in a case of cancer I mentioned at the Medico-Chirurgical Society of Edinburgh on 4th June 1890, but I said that I did not know whether they were special nuclei or a foreign organism.

This, of course, led to further investigation, and to the examination of other cases of cancer, with the result that the structure was found in all those examined. For laboratory use it was necessary to have a convenient name for these, so I called them fuchsin

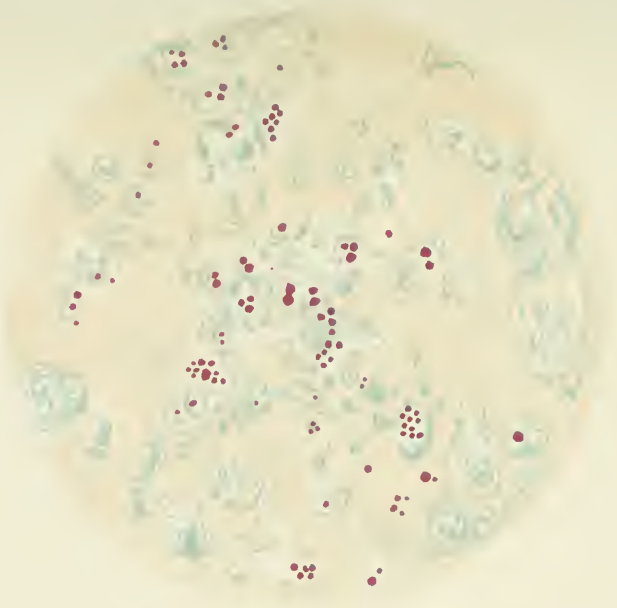


FIG. 1.

Showing "Fuchsin Bodies" singly and in groups, mainly in epithelial cells, each group surrounded by a clear space. From cancer of adrenal. Stained by special method (see letterpress). (Pillischer, oc. 3, ob. ⅛ × 285).

A. Macdougall, del.

Drawn on stone and printed by W. & A. K. Johnston, Edinburgh.

bodies, and this name I propose to adhere to until their relations to cancer and their biological status is determined.

With this discovery all kinds of possible error were suggested to my mind: Were they accidental impurities in my material, bottles, or stains? Was it a mere piece of staining legerdemain? Were they the nuclei of tissue cells in exaggerated formative and reproductive activity? Or were they simple globes of some form of degeneration? All these questions I set myself to answer. Tissues from the same bottles, preserved in the same fluid, and cut at the same time, were examined without any indication whatever of accidental contamination. The idea of staining legerdemain was excluded by the impossibility of producing the effect in

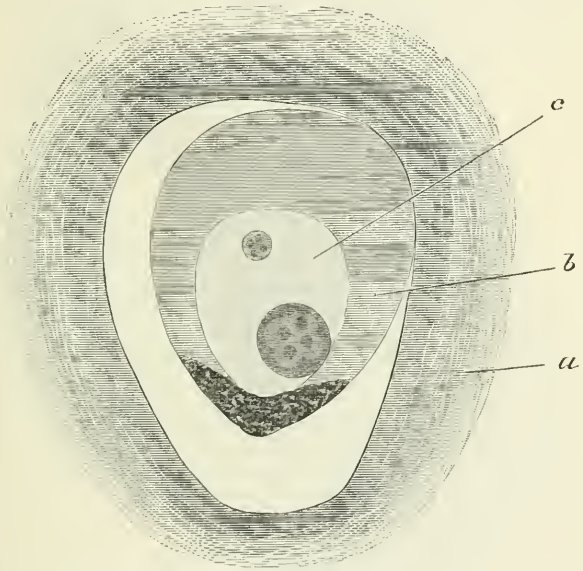


Fig. 2.—Large epithelial cell *b*, in space surrounded by fibrous tissue *a*, and showing a lower part nucleus; *c*, vacuole containing two organisms.

non-cancerous parts. Although from their perfect roundness and homogeneous hyaline-like structure it seemed impossible they should be nuclei, still the remote possibility had to be dealt with. Organizing inflammation of serous membranes showed that the nuclei of the formative cells did not give the reaction, neither did the cells in tubercle of the lungs, in typhoid lesion of the intestine, in inflammatory affections of the meninges, lungs, etc., in granulations, nor in the organs of an embryo at the fourth month. Then as to degenerations: fatty degenerations and infiltrations, waxy degeneration in different organs, colloid goître, myxoma, myxomatous sarcoma, degenerative changes of epithelium as in tubular nephritis, spinal cord degenerations, and, in

fact, all kinds of morbid material which I could think of, were examined, and, with the exceptions I shall refer to presently, with a like negative result. Then as regards their presence in other tumours, the sarcomata were examined early, and gave negative results, although in one case in which there were extremely large cells, and which I thought might be an unusually large-celled sarcoma, the bodies were found, and their presence, I think, probably indicates that the original view I was inclined to take of this growth was wrong. In simple tumours, such as fibromata,

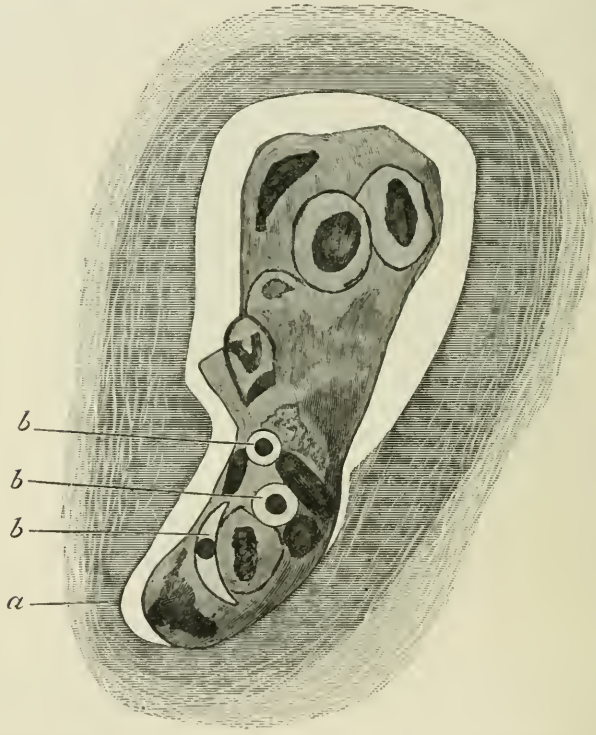


FIG. 3.—Mass of epithelium lying in alveolus wall (*a*), formed of fibrous tissue; *b b b*, encapsuled organism.

papillomata, myomata, etc., they were not found. In venereal warts and condylomata they were not found, nor in primary syphilitic sores, nor in the ulcerated tissue and crust of a syphilitic skin affection. A tumour taken out of my practical class material and labelled "adenoma of mamma," and which is very rich in adenomatous structures, showed the bodies. A tumour of the dura mater from the same material, which has been in my possession for the last seven years, and which I think is certainly a gumma, showed the lymphatics in its neighbourhood to contain numbers of these fuchsin bodies; of this case, as well as the preceding, I at present

know nothing. Recently I had a syphilitic case in the post-mortem room, which was both exceptional and extraordinary—a case in which, some six or seven weeks after primary infection, there was not only a skin eruption, but extensive destructive lesion of the fauces and larynx, and even of the bones of the vertebræ behind the fauces. This case had absolutely defied treatment. In the larynx of this case I found a few fuchsin bodies. One other tumour I may mention—an aural polypus sent over for report from the Throat and Ear Department. I found this polypus to be in its greater part fibro-myxomatous in structure, but in its deeper part adenomatous, and in this adenomatous part I found a few fuchsin bodies. I wrote to Dr M'Bride asking him if he had any suspicion of its being malignant, and he replied that he did not know, but that it was no sooner removed than it

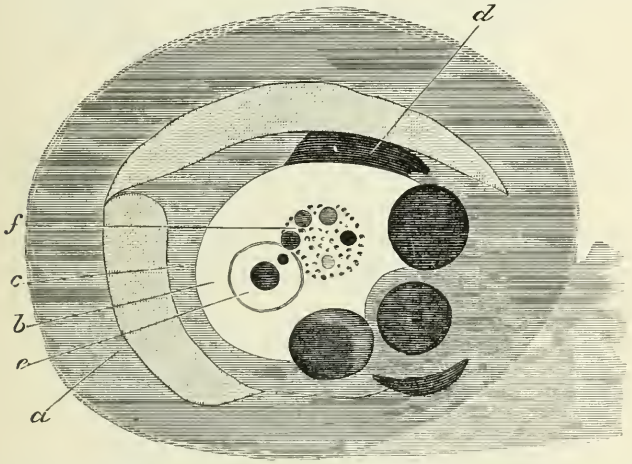


FIG. 4.—Wall of alveolus, *a*; *b*, large vacuole surrounded by remains of epithelial protoplasm, showing at *d* nucleus; *e*, encapsuled organism lying in vacuole; *f*, degenerating organism, showing spores in its interior.

commenced to grow again, and that he could not get the patient to come regularly to have it attended to. In three cases of gelatinous degeneration of the knee-joint examined they were found in one, and this case had, I believe, old sinuses. In a subject in the post-mortem room, with a large ulcer on the leg with a large island of skin in the centre of it, I found a few fuchsin bodies in one section, but could not find them in any other sections. Altogether tissues have been examined from fifty to sixty different cases, sometimes four, five, or six sections of the same tissue selected with the determined purpose of subjecting the positive observations to the severest possible tests. The result has been that fuchsin bodies were found in one case of chronic ulcer of the leg, one of tuberculous disease of a

joint with old sinuses, one of phenomenally severe destructive and intractable syphilitic lesion. These were cases of which I knew

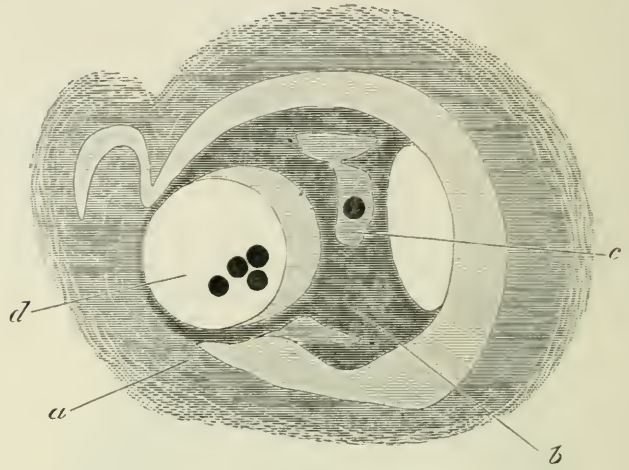


FIG. 5.—Fibrous wall, *a*, containing mass of epithelium, *b*; *c*, nucleus with parasite in interior; *d*, vacuole with four fungi.

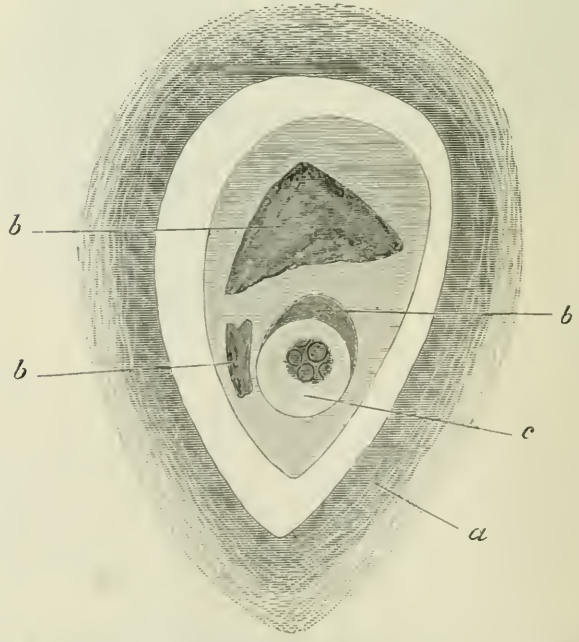


FIG. 6.—Mass of epithelium in alveolus, showing nuclei at *b b b*; *c*, vacuole containing degenerating organism.

something. Then there were two cases of which I have at present

no record, one a case of mammary adenoma, and one a gummatous tumour of the meninges. I need not dwell upon the possibility of ulcerated free surfaces becoming contaminated by organisms, nor need I do more than remind you that chronic ulcers assume at times malignant characters. With regard to their presence in the remarkable case of syphilis to which I have referred, and also in a gumma of the dura mater, I am at present content to repeat that in one of these there was a phenomenal destructiveness and intractableness, and in the other probably a like intractableness to treatment, and to indicate the possibility of there having been a dual infection. At all events, I think you will agree with me that a more severe set of check observations could not have been selected, and the occurrence of the organism in the exceptional cases mentioned could not be regarded as sufficient to overthrow our other evidence. In fact, to my mind, they but suggest possible solutions of various phenomena which have been recognised but hitherto not explained.

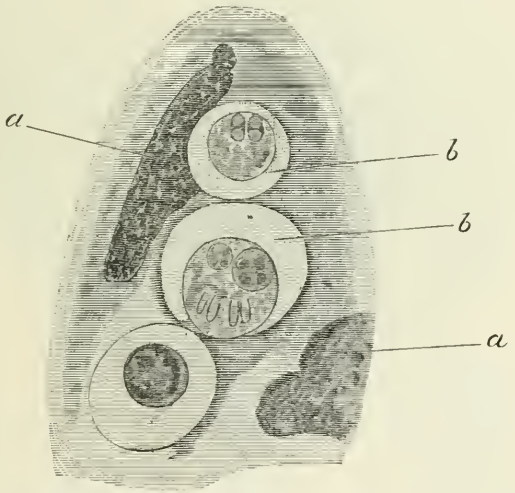


Fig. 7.—Mass of epithelium showing nuclei at *a a*; *b b*, vacuoles containing degenerating organism showing spores.

So much for the check observations and the negative side; and now I turn to my cases of cancer and the positive side. Forty-five cases have been examined, and that there are not more is simply due to want of time. These were taken either because they happened to be cut and ready for examination, or as they were sent over fresh from the surgeons, or in chronological order out of my hospital material. They include malignant epithelial growths of very varied structure, as epitheliomata of the lips, face, and antrum, rodent ulcer, scirrhus of the mamma both primary and recurrent, a spreading papilloma of the foot for which ampu-

tation had been performed, a malignant nodule in the foreskin, malignant adenoma of cervical glands, cancers of the stomach, liver, spleen, abdominal glands, supra-renal capsules, uterus, and ovaries—material from forty-five separate individuals, not forty-five affected organs, for in some several organs were affected. One of these cases was a very remarkable one, the pathological position of which is still uncertain ; another was represented by sections in a bottle labelled "epithelioma" and dated 1885—a time when my pathological material consisted of odds and ends. In these two no fuchsin bodies have been found, but in the remaining forty-three they have. As regards number, they vary greatly in the individual sections cut at the same time from the same bit.

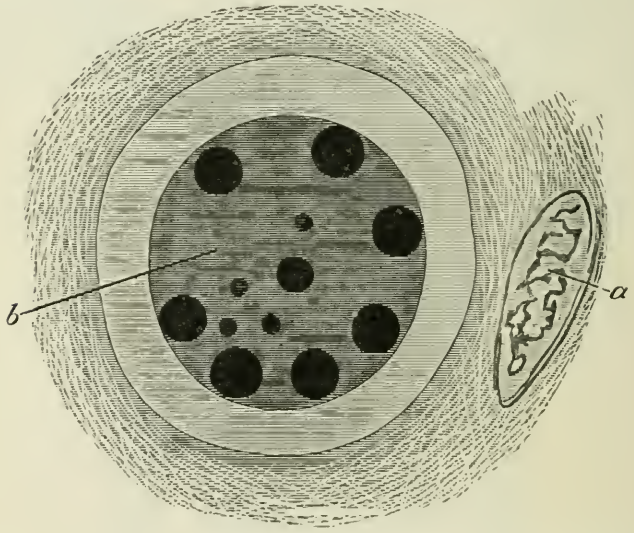


Fig. 8.—Large degenerating organism (*b*) showing spores; (*a*) nucleus of epithelium.

This was very forcibly illustrated in a bottle of sections of a cancerous adrenal, in which they were present in great numbers in certain parts of the sections. It was, indeed, this case which gave me the clue to the nature of these organisms, as will be seen presently. They were so numerous and so unmistakable in these sections that they were used lavishly for all kinds of staining, bleaching, counterstaining, and comparison, until they were exhausted, and three other pieces of the same adrenal have been cut, the sections from any of them showing only a few groups in each. The special abundance of the bodies in foci was noted in other cases, and may possibly occur in all. I must also say that they are not necessarily present in every section, nor are they necessarily present in every piece of a tumour which may be cut, although we have seldom had to cut two pieces to find them.

As regards their distribution in the various constituents of the morbid growth, they may be present in the small-celled infiltration at the margin of a cancer, or amongst and in the epithelial cells in the

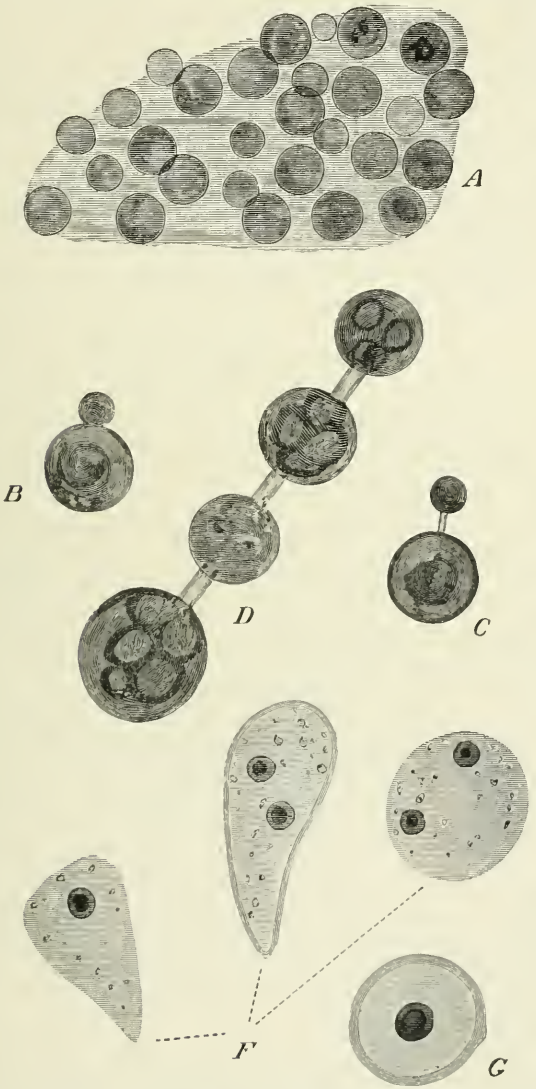


Fig. 9.—A, mass of fungi; B, individual giving off bud; C, same, but bud further removed from parent but still attached; D, four individuals attached to one another; E, four individuals attached and overlapping; F, small spores in lymph cells and leucocyte; G, altered leucocyte containing spore.

cancerous alveoli, as in Fig. 1; or in the stroma, or in the lymphatics. In one case of very diffuse infection of mammæ, liver, and spleen,

etc., I found them in the spleen beyond the malignant nodules. I have also in at least one case seen them in the small-celled infiltration in a portal space in the liver beyond the cancer nodule.

As a rule, which is comparatively rarely departed from, they occur in little clusters or groups of two, three, four, five, up to twenty or more. Wherever they occur they almost always show a clear space round them. They can be found readily with a lens of 100 diameters if the light is good, and the search is facilitated by the use of an Abbe's substage condenser. Their brilliant red or purplish red colour forms a striking contrast to the green and delicate purple of the tissues, as seen in the coloured figure. The individuals of which the groups consist are in form perfect spheres. They vary greatly in size, the largest being about 12μ in diameter, that is, half as large again as a red blood corpuscle, others are 11, 10, 9, 8, 4μ, and even much smaller, the commonest size being 4μ. They appear perfectly homogeneous and structureless as examined by daylight,[1] and the larger clumps are held together by a delicate cementing substance which stains faintly. Such are the observations which even an inexperienced microscopist will have no difficulty in making if he has succeeded with the differential staining.

I must not leave this part of the subject without referring to another complication which troubled me greatly for some time, and that was that the nucleus of the cancer cells retained in some instances the fuchsin dye. This was all the more confusing as the nuclei of the cells in the unaffected tissue did not do so; however, by a process of bleaching the colour could be turned out of the nuclei while the bodies in question still retained it. In the meantime I cannot more fully dwell on this point, although it includes some exceedingly interesting and important phenomena, which must remain for future consideration.

As it seemed to me, there was no escape from regarding these structures as special organisms which—so far at least as my pathological material was concerned—were practically confined to cancer, the question was, What were they? Were they animal or vegetable? and what was their mode of growth and reproduction? Before attempting to answer this question it is necessary to look at the work which has been done, especially on the Continent, in the study of cancer, and the contentions which have been based upon that work.

The bacilli found in cancer by Scheuerlen,[2] Verneuil, and Koubasoff may here be passed by, to enable us to reach at once the work which describes a parasite, belonging to the lowest subkingdom of animals, as occurring in some cases of cancer. In this

[1] Their internal structure requires artificial illumination for its determination, and it is not dealt with at present.

[2] "Die Pathologie des Carcinoms," *Deutsch. Med. Woch.*, 1887, p. 48.

connexion it is necessary to point out that the first epithelial growth in man in which a parasite of this kind was held to stand in causal connexion was, so far as I am aware, molluscum (or epithelioma) contagiosum. Virchow pointed out the resemblance of certain structures in this disease to gregarinæ, but Bollinger[1] is entitled to the credit of having more definitely asserted this, owing to the resemblance between this disease as occurring in man, and a similarly named disease in fowls which was specially studied and described by him

Professor Neisser,[2] of Breslau, published in 1888 an elaborate paper on this subject containing the results of his own observations, in which he places the parasite in the coccidia group of the sporozoa. The drawings he gives in support of this contention I need not specially refer to, as I am not in a position to form an opinion on them, not having had the opportunity of studying this disease. I may, however, say that I am by no means convinced of his contentions by the figures given.

Last year (1889) two communications were made to the Société de Biologie by Darier, working in Malassez's laboratory, and seemingly inspired by him.[3] In the first of these,[4] communicated on 23rd March, he intimated the recognition of a coccidium in a case of *acné cornée*, and defines the condition as a *psorospermose cutanée*; in the second, communicated on April 13th,[5] he intimated that he had found a parasite belonging to the same class in a case of Paget's disease of the nipple. He says that they present all the degrees or stages of evolution of these organisms; at first a naked mass of protoplasm, afterwards surrounded by a membrane, then dividing into very numerous granules (*grains*) contained in a cyst; and he infers that the disease is a parasitic one, a *psorospermose*. He gives no figures.

To the same Society, on 6th April 1889,[6] M. Albarran intimated that he had recognised organisms of a like nature in two epithelial tumours of the jaw. He at the same time mentions that M. Malassez had observed analogous forms in many tumours. He also gives no figures.

In the *Fortschritte der Medicin*[7] of 1st June 1889, Professor Thoma has a very short note, without illustrations, on "A Characteristic Parasitic Organism in the Cells of Carcinoma." He describes it as a unicellular organism, consisting of protoplasm and a nucleus, with sometimes a nucleolus. They vary in shape, being round or

[1] Virchow's *Archiv*.

[2] *Vierteljahresschrift für Dermatologie und Syphilis*, 1888. Retzius also has a paper which I have not seen; Om. Mollus. Contag., *Nord. Med. Ark.*, 1870, ii.

[3] See *Arch. de Méd. Expérimentale et d'Anatomie Pathologique*, 1st March 1890, p. 302.

[4] *La Semaine Médicale*, 1889, p. 101.

[5] *Loc. cit.*, p. 125.

[6] *Loc. cit.*, p. 117.

[7] *Fortschritte der Medicin*, No. 11, p. 413, 1st June 1889.

oval. They are present singly or in groups in the nucleus, the latter becoming vacuolated. In other cases the cyst is near the nucleus. He says there is a strong temptation to regard these as encapsuled coccidia, but this interpretation is still doubtful.

Louis Wickham, on 1st January 1890, published a long and interesting paper[1] on "The Pathological Anatomy and the Nature of Paget's Disease of the Nipple," in which he describes and figures appearances which he regards as coccidia or psorospermiæ.

Professor Klebs published, in June of this year,[2] papers "On the Nature and Diagnosis of Cancer Formation," in which he discusses these questions with fairness and masterliness. In them he refers to hyaline bodies present in cancer, which, however, he is decidedly disposed to regard as degenerative products. It is not altogether clear what he means by his hyaline bodies, for he speaks of them as present in the inner parts of the proliferating epithelial tubes, which were filled by them, partly in a rounded, but mostly in an angular form; they were also present in the stroma, but more sparsely. The figure he gives does not help us to form an opinion on the nature of the structures to which he refers. I am, however, disposed to regard most of his hyaline bodies as productions of the cells, for hyaline masses are frequently present, and are easy of recognition in the alveoli of the more adenomatous cancers.

Then, lastly, in July of this year, Von Nils Sjöbring[3] describes a "Parasitic Protozoa-like Organism in Carcinoma," of which he gives figures, and which he found in six cases of cancer of the mamma. He follows it from a simple cellular stage to the stage of spore formation.

Summing this up, and leaving out any further reference to molluscum contagiosum, we find that Albarran, Darier, Thoma, Wickham, and Sjöbring have found in cancer what they believe to be an organism. All of them, with the exception of Thoma, describe their organism as belonging to the protozoa, while Thoma does not commit himself; and, as has been said, only Wickham and Sjöbring give figures to aid us in forming a judgment on their contentions.

With the object of elucidating this subject, I may be permitted to refer briefly to the lowest sub-kingdom of the animal world, which is divided by Leuckart into three classes,—the rhizopoda, the sporozoa, and the infusoria. The sporozoa contain, according to Balbiani,[4] five groups or orders,—the gregarinæ, the psorospermiæ oviformes or coccidia, and three other groups of psorospermiæ. Of these the gregarinæ occur as parasites in the invertebrates, while the psorospermiæ occur in the vertebrates. As regards the

[1] *Archives de Médecine Expérimentale et d'Anatomie Pathologique*, 1889, p. 46.
[2] *Deutsche medicinische Wochenschrift*, Nos. 24, 25, and 32, June 1890.
[3] *Fortschritte der Medicins*, No. 14, 15th July 1890.
[4] *Leçons sur les Sporozoaires*, Paris, 1884; see also *Die Protozoen als Krankheitserreger*, von Dr L. Pfeiffer, Jena, 1890.

latter group, it is important to understand their structure and development. They are described by the authorities as unicellular organisms covered by a more or less firm capsule or shell, which latter, in the coccidia, have a double contour, and the contents either fill the shell or are gathered into a rounded mass. Reproduction in them all is by means of spores (pseudonavicellæ or psorospermiæ) formed in the interior of the adult, and in these spores are developed sickle-shaped bodies which escape from the spore and become new parasites.[1] This mode of reproduction is an essential factor in the determination of the biological position of these parasites. To meet this necessity, Sjöbring has figured the spore formation in the cancers he examined.

Wickham's organism consists of a double contoured capsule either filled with protoplasm, or the protoplasm is gathered into a mass in the centre. He does not give any figures of the formation of spores as occurs in typical psorosperms. In fact, looking at the work on this subject in the concrete, I regard some of the figures as having nothing whatever to do with foreign organisms, for I am familiar with the appearances represented; others have certainly been misinterpreted, while some figures probably represent the organism with which I am dealing.

To return now to the consideration of our fuchsin bodies, we have seen that they occur usually in groups, the individuals of which vary greatly in size, but can be seen with the ordinary working lenses magnifying from 300 to 400. For more detailed investigation, I have worked with a No. 7 objective of Leitz and one-sixteenth oil immersion by Reichert, and a No. 3 or No. 5 eyepiece. They may be studied as stained by the special method given here, or by logwood and eosine, or by Gram's method with methyl-violet. With reference to logwood, I may say that it does not stain the bodies under consideration, but they are tinted with eosine. Each group, and most of the isolated individuals, is surrounded by a clear area, which clear area has often the appearance of being bounded by a definite capsule. This appearance is brought out in Fig. 1, *et seq.*, and the study of the isolated fuchsin bodies is necessary for the complete understanding of this interesting link in the history of the organism. What I find is this: An isolated individual is present, for example, in an epithelial cell, as in Fig. 2; the cell protoplasm is stained faintly with logwood, and the nucleus is deeply stained, while an eosine-stained globe is present in the cell protoplasm, the globe being surrounded by a clear area or vacuole, which has such a definite limit that it looks somewhat like a capsule, but it can be seen by focussing that the free edge of the naked epithelial cell gives an exactly analogous effect, so that I have no hesitation in saying that there is no true capsule. As regards the clear space itself, while in many places it looks as if it were empty, in others it contains a structureless, very transparent

[1] Leuckart, *The Parasites of Man*. English Translation.

substance which tints very delicately. A similar structure is to be found in masses of epithelium (Fig. 6) welded together and lying in spaces. But, both in these epithelial masses and in the vacuoles referred to, there may be small fuchsin bodies surrounded by a clear space and bounded by what we must call either a capsule or a limiting structure. These are represented in Figs. 3 and 4, and their mode of formation has to be dealt with. Now these appearances are of great importance, for on them might be based the contention that here we have to do with an encysted sporozoon, coccidium, or psorosperm. Next, it is to be noted that in the vacuoles there may be two or more small fuchsin bodies (Figs. 2 and 5); in other parts the fuchsin body has become granular, lost its characteristic staining reaction, but shows in the

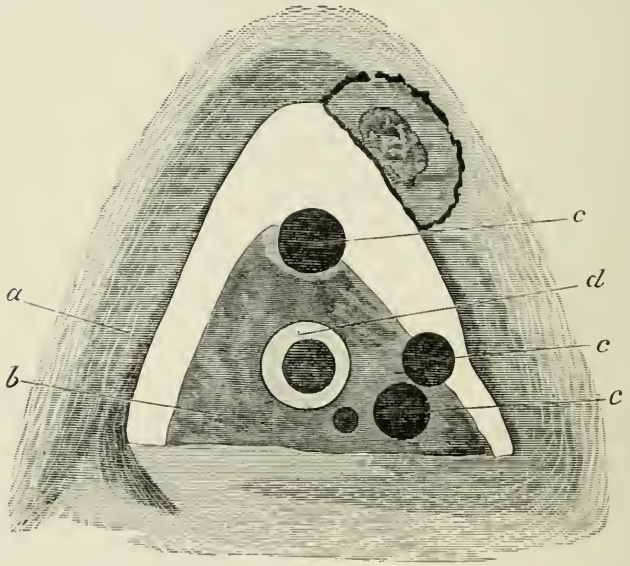


Fig. 10.—Large epithelial cell (*b*) containing spores (*c c c*) without vacuole, and (*d*) spore with vacuole.

midst of the granules minute eosine-tinting bodies undoubtedly spores (Figs. 4, 6, 7). Further, I draw attention to Figs. 6 and 7; in them the fuchsin bodies, by our own staining method, are coloured purple instead of red; and this purple colour gave me trouble in my earlier investigations. I then excluded all purple-stained structures, and there is still caution required in the admission of some of them. In these, as the organism becomes granular and degenerates, the spores in it become more visible. In Fig. 8 there is a very large fuchsin body stained purple, and lying in its space, and in it spores are very distinctly present. In Fig. 5 there is a large space with four free purple-stained fuchsin bodies, and in the nucleus of an adjoining epithelial cell a similar

body which, I take it, has recently migrated; and it is to be noted that this has no vacuole round it. A similar appearance is seen in Fig. 10 (logwood and eosine), where, in a large epithelial cell, there are four fuchsin bodies not surrounded, while one is surrounded by a vacuole.

From all this it might still be contended we were dealing with a protozoon, but I have to draw attention to Fig. 9, from a section stained by Gram's method with methyl-violet. This stain shows the process most diagrammatically, although it is to be observed by other staining methods. Here it is quite unmistakably to be seen that a large fuchsin body gives either off or out a small globular body (Fig. 9 B), which gradually increases its distance from the parent body, but remains attached to it by a delicate filament (Fig. 9 C); this bud grows and gives off another, and so on, and there is obtained such a figure as is represented in Fig. 9 D. In other cases, however, there are rows or clumps, the individuals of which overlap one another, as seen in Fig. 9 A, or a large fuchsin-stained body shows arcs of circles projecting from it. Further, by Gram's method smaller spores can perhaps be seen than by other staining methods, and some appearances are exceedingly suggestive of a parent body having vomited out a number of minute spores. By this same method of staining it can be seen that the small spores appear in the lymphoid cells or leucocytes of the infiltrated area (Fig. 9 F), that the effect of this entrance is that the cell-protoplasm becomes clearer, and the chromogenic granules are driven to the circumference of the cell; at all events they disappear, with the result that the small fuchsin body is surrounded by a clear space, with a distinct limiting ring formed by the remains of the comparatively unaltered protoplasm (Fig. 9 G). This, I take it, is the mode of formation of what we may call the encapsulated fuchsin bodies, lying in vacuoles or amongst epithelial cells, as in Figs. 3 and 4. Now, as to the appearances in the interior of the epithelial cells. When the fuchsin body first enters them no vacuole is present (Fig. 10), but a vacuole is produced (Fig. 10 *a*), this vacuolation being evidently simply a change wrought in the cell protoplasm by the fuchsin body which leads to its clarification, increased transparency, and to a loss of tinting capacity. The limit of this change is a definite line, as distinguished from a gradually shaded line, and thus the appearance, which might be mistaken for a capsule, is produced.

From all this there is in my mind absolutely no doubt that the organism here is a fungus which belongs to the sprouting fungi (*Sprosspilze* of Nägeli).[1] The proof of this is by no means to be readily found in every section nor in every case, for the usual arrangement—as demonstrable by the fuchsin and iodine green method—is that of clusters. The explanation of this, I think, is

[1] *Die niederen Pilze*, München, 1877. See also *Fungi, Mycetozoa, and Bacteria*, by A. de Barry, English translation, 1877.

that our method of staining acts best when the organism is at a certain stage of its growth, and that the smallest spores and degenerating larger individuals either do not stain differentially, or they stain purple from a combination of the two colours used.

In conclusion, it is only necessary for me to remind you that this class of fungi includes the yeast fungus, and that if the presence of this parasitic fungus in cancer is confirmed by other observers, we have found in it an organism which from its very character implies the production of a fermentation product; while the nutrition, the reproduction, and the death of the fungus cannot be conceived as occurring in the tissues without producing changes not disproportionate in magnitude to the anatomical changes present in cancer.

I wish further to take this opportunity of acknowledging the loyal devotion and the untiring industry and zeal, as well as the technical skill and care, with which my friend and pathological assistant, Mr W. F. Robertson, has helped me in this investigation.

The President remarked on the singular conjuncture of their having before them on the same evening two papers of such outstanding interest as those of Dr Philip and Dr Russell. It might well be that the meeting should prove to be of historic interest in the progress of Medicine. If the mortality from consumption became markedly lessened through Koch's treatment, the mortality from cancer, great enough already, would become all the greater. The first step towards the discovery of what promises to be a cure for consumption was taken when Koch discovered the microbial cause of the disease and ran to earth the tubercle bacillus. If their able Secretary, Dr Russell, had really discovered the micro-organism that causes cancer, then the first step had been taken towards the scientific search for an effective remedy. It was, perhaps, too late to enter that evening on a full discussion of this important communication. At the same time there were numbers from a distance who might not be able to be present at a subsequent meeting, and if Professor Gairdner or others were disposed to offer any observations, the Society would be glad to hear them.

Mr Stiles said he should like to be allowed to congratulate Dr Russell upon the important and admirable paper which he had listened to with the greatest interest. He had of late examined a considerable number of cancers, and possessed drawings of bodies which he was inclined to look upon as of parasitic nature. His sections had been stained with carmine and hæmatoxylene, but he believed they were similar to Dr Russell's "fuchsin bodies." Dr Russell's admirable and successful work with the aniline dyes went far to convince him of their parasitic nature.

On the motion of Dr Bruce, the discussion was adjourned.

Meeting III.—December 17, 1890.

Mr A. G. Miller, *Vice-President, in the Chair.*

I. Discussion on Dr Russell's Paper.

The discussion on Dr Russell's Paper on the characteristic organism of cancer was then resumed.

Dr Bruce referred to the great interest which had been excited among pathological workers in Edinburgh by the valued contribution of Dr Russell at their previous meeting, and stated that he felt sure that whether or not the members of the Society saw their way towards agreement with Dr Russell's views, they must none the less set a very high value upon his work. In the remarks which followed he would prefer to point out possible sources of error in the observations which would require to be eliminated before these observations could be accepted as conclusive. It was necessary, before accepting Dr Russell's conclusions, that they should be satisfied that the differential method of staining which he employed selected only bodies of the nature of organisms. It was remarkable that the bodies which retained the fuchsin dye were of extremely various sizes,—a fact which rendered it very unlikely that they should be organisms; and, further, the relationship which these bodies showed to each other, as illustrated by Dr Russell's sections and diagrams, was not at all that of any known yeast plant. In one section, which was figured in the *British Medical Journal*, a rounded homogeneous looking body in the interior of the nucleus had retained the fuchsin dye, and it appeared more probable that these represented the chromatin of the nucleus gathered into a mass in the centre of the nucleus rather than that they represented organisms. On staining a series of sections from a carcinoma of the liver, Dr Bruce had seen all stages of transition between the chromatin network filling the nucleus to the rounded mass of pigment in the centre of the nucleus. Other bodies within the epithelial cells might be, and very possibly were, degenerated leucocytes which had been absorbed into the cells and partially digested there, or represented forms of local degeneration of the protoplasm of the cell. In the cells in the connective tissue in the neighbourhood of the tumour it was possible that the fuchsin bodies around the nucleus represented granules of degeneration similar to, if not identical with, those which Ehrlich had found to absorb eosin with great readiness. If such cells were to break up, the granules would appear as free minute spherules with a remarkable resemblance to micro-organisms, but of this matter others would probably speak who were more conversant with the subject. Dr Russell had apparently not endeavoured to cultivate these bodies, which was still the only satisfactory proof that they

were of the nature of organisms. It was further to be noted that the existence of these fuchsin bodies was, as Dr Russell himself confessed, not limited to carcinomata, but that they were found in certain other growths which could not in any way be regarded as of that nature. In conclusion, Dr Bruce expressed his admiration for the beautiful work involved in this painstaking research, and hoped that Dr Russell would continue his observations until he had satisfactorily demonstrated that he was dealing only with living organisms.

Dr Gulland regretted that he was unable to agree with Dr Russell's views. He had experimented extensively with the fuchsin staining method in cases of cancer, and had not succeeded in finding anything in the tissues so stained that he could not explain from a histological standpoint, without calling in the aid of micro-organisms. He found that the following structures retained the fuchsin,—Cornified epithelium, some red blood corpuscles, the nuclei of degenerated leucocytes, nucleoli of cancer cells, especially when these were degenerated, and possibly fat globules (*cf.* Gottstein, *Fortschritte der Medicin*, 1886, p. 252). These were comparatively unimportant, but he found that the granules in the cells called by Ehrlich "Mastzellen" or "basophile leucocytes" gave exactly the reactions described by Dr Russell as characteristic of his "fuchsin bodies" (*cf.* Friedländer, *Mikroskopische Technik*, p. 107), and were, moreover, found in the situations where Dr Russell found his organisms, and he thought that Dr Russell must show clearly that the two had no connexion before his conclusions could be accepted. He would point out, moreover, that there was a great difference in size and arrangement between the bodies stained by fuchsin and those demonstrated by Gram's method, and he had some reason to believe that these latter bodies were not identical with the fuchsin bodies, but were due to an imperfection in the method of staining. He failed also to understand why Dr Russell had placed his organism among the yeast-fungi, as neither the staining reaction, the structure, nor the budding figured were those characteristic of torulæ, and he could not understand how cells of the comparatively low vitality of yeast-cells could act on human tissues so powerfully as to produce cancer, or could resist the attacks of the leucocytes, among which Dr Russell found so many of his fuchsin bodies.

Prof. Chiene said that much interest had been taken by the profession in Edinburgh in Dr Russell's paper, and that in the Surgical Laboratory of the University carcinomata had been examined with a view to test whether or not the fuchsin bodies were organismal. Mr Edington, who had, at Mr Chiene's suggestion, investigated the subject, had reported "that the bodies were non-organismal, and in all probability were a degeneration of the nuclei and protoplasm of epithelial cells of a hyaline nature." The whole subject was one of much interest, and further work was required to settle their exact nature.

Mr Cathcart had many difficulties in accepting Dr Russell's "fuchsin bodies" as organisms characteristic of cancer, or in fact of accepting any organisms as the cause of cancerous growths. Like Dr Bruce, he had been struck by the varying size of these fuchsin bodies, and had, moreover, noticed as remarkable the uniformity in size of members of the same group, while the size was so variable among the members of different groups. If this were an organism which had the peculiarity of appearing in such varying sizes, it was strange that these presumably developing cells of the same age should so often remain together, and in groups of different numbers. Another difficulty was that only one species of organism was supposed to be the cause of many different kinds of cancer. This was a difficulty, because among epithelial tissues there was probably a greater variety of "soil" than could be found in any other class of tissues. On the other hand, if we were to grant this single organism as the possible cause of various cancers, two other difficulties came in the way, which were worth considering,—(1.) Why it should happen that cancers seemed always to spread from the first centre of outbreak? If an organism capable of setting up mischief in any epithelium were present in large numbers in a patient's body, it was remarkable that we never had in the last stages of the disease an outburst of different forms of cancer, but always secondary deposits distinctly traceable to the first. (2.) Why these epithelial organisms should, when on their way into the blood, so often pass through without affecting the epithelial covering of skin and mucous membrane, and afterwards settle down in some ductless gland or the outlying lobule of an atrophying mamma? As to the possibility of cancerous growths being caused by micro-organisms, the more he considered it the more improbable it seemed to be. The arguments in favour of the organismal view might be summed up in this, that both malignant tumours and infective granulomata were infective in the patient's body, locally, by the lymphatics, and by the blood. Should future experiments, more successful than those hitherto, show that malignant tumours, like most infective granulomata, were contagious from one individual to another, that result would only widen the infective range of the former, not prove that the cause of the infectiveness was the same in both. On the other hand, he thought that the argument from analogy in structure and clinical history became weaker the more closely it was examined. In infective granulomata it was quite possible to trace irritative changes only differing from those produced by mechanical and other injuries by certain degrees explicable on the ground of the varying character of the different organismal poisons. In cancerous growths, however, the phenomena differed from those of irritative changes in epithelial texture so much that it seemed reasonable to look for a cause different from irritation even of a peculiar kind. For instance, the invading character of an epithelium seemed to be

something quite different from the outgrowth and overgrowth of the same tissue in response to irritation. Again, in secondary cancerous growths the preservation of the character of the original tumour was a feature which had no analogy in the secondary deposits caused by infective organisms, where the appearances could be traced to organisms only and to their peculiar modes of irritation, as already noticed. In cancerous secondary deposits, therefore, if organismal in origin, we must presuppose the transference not only of the micro-organism, but of formative cells to be stimulated by that organism. There was nothing impossible in this, but it had no analogy in what was already known of infective organismal growths. Lastly, an examination of the structure and clinical history of cancers seemed to lead insensibly into sarcomata on the one hand and into adenomata on the other. If this view of gradation were admitted—and it was admitted by many who yet expect that a parasitic origin will ultimately be found for malignant growths—we would have to show at what point in the series the micro-organisms step in, or by what modification of our previous knowledge they would account for, say, a chondro-sarcoma of the testis.

Dr Barrett had not yet had an opportunity of thoroughly testing the action of the stains employed by Dr Russell, but was acquainted with bodies having the same microscopic characters when stained by Gram's method. In these, not only groups corresponding in size to fuchsin bodies, but some also showing an arrangement in rows, with a faint or almost unstained narrow band uniting them, similar to the diagram in which four fuchsin bodies are shown united by a slim stalk or band. The cases in which these bodies had been found were,—1st, A case of advanced atheroma of the aorta, which was a simple case, uncomplicated by any malignant disease ; 2nd, In the kidney in a case of nephritis. In these cases the bodies resisted decolorizing agents very strongly after staining in aniline dyes. With regard to their nature, they were not calcareous particles, nor were they ordinary hyaline degeneration of cells, inasmuch as hyaline material behaves very differently with aniline stains. Further, in these specimens they were not the degenerated nuclei described by Friedländer, nor are they the basiphiles mentioned by some of the speakers. Fat particles they certainly were not in the atheromatous case, as when stained with osmic acid they did not react to it. They were probably a form of pigment derived from blood, possibly an intermediate stage in pigment formation.

Dr Macfadyen avowed his belief that the etiology of carcinoma was not to be sought in a micro-organism. It was impossible to draw a distinct line of separation between the carcinomata and the adenomata, or between the latter and the teratomata, and the microscopic theory could not possibly account for the genesis of either the adenomata or the carcinomata. He was not able on the

evidence adduced to accept Dr Russell's fuchsin bodies as vegetable organisms. He pointed out that these bodies had been very fully described by Cornil and Alvarez as occurring in the lesions of rhinoscleroma; but these authors had regarded them as degeneration products of the cell protoplasm. He suggested to Dr Russell that he should try the effect of a solution of caustic potash on the bodies, as that would be the simplest method of distinguishing between products of cell degeneration and such vegetable organisms as the torulæ. Assuming that these fuchsin bodies were micro-parasites having an etiological relationship to the carcinomata in which they were found, it ought to be easy to furnish conclusive evidence of this connexion. It was impossible to believe that cancer was transmitted by contagion, and they must therefore assume that the organism was not an obligatory parasite, and it ought therefore to admit of cultivation outside the body. Moreover, to his own knowledge, cancer occurred spontaneously in all the common domestic animals save the sheep, and there ought, therefore, to be no difficulty in transmitting carcinoma experimentally.

Dr Haycraft thought that the subject had been discussed with insufficient data. Even the chemist had to apply test after test before he could be certain of the identity of a mixture under investigation, and the same remark applied with even greater force to the work of the histologist. A single test—that of fuchsin, for example—must first be combined with many other staining reactives, and, above all, the solubility of the globules in acids, alkalies, boiling alcohol, ether, etc., must be carefully investigated before they were certain that they were dealing with any specific structure. Afterwards the statistical method was capable of giving a definite answer as to whether these bodies were to be associated with cancerous growths, and with these alone.

Dr Russell referred in detail to various difficulties which had been suggested as to the retention of fuchsin in horny epithelium, in degenerating red corpuscles, by fat, and to changes in the chromogenic granules in cells, and said that the retention of fuchsin by irregular portions of protoplasm in epithelial cells and by some nuclei was a great source of trouble at first, but by various bleaching methods the colour was turned out of these, while the bodies in question retained the colour. But with reference to this subject, there were many appearances of great interest with which he hoped at some future time to deal. All the difficulties suggested on these points during the discussion had suggested themselves already to his mind, and he considered he had overcome them. As to the difficulty of accepting an organism as the cause of cancer, the difficulties suggested were much on the same lines as the objections raised in all instances where an organism had been suggested as the cause of a special disease. He was not prepared to explain how the bacillus of tubercle found

entrance, for example, to a knee-joint without external opening; neither was he prepared to say how this organism found its way to some equally out-of-the-way spot. Then as to the different forms of cancer, he thought many of our present ideas required to be reconsidered. As to the nature of the organism in question, the greater number of his figures were produced to bring his work into line with work on cancer which had been done in France and Germany, and might have been altogether omitted, and it was quite possible that some of these might require correction. Referring to the objections to the structure under consideration being a member of the sprouting fungi, he again drew attention to the appearance of bud formation and the attachment of these to the parent, and the continuation of this budding process to the formation of clusters and groups, and that notwithstanding there might be differences as compared with the better known unicellular fungi, still the process was more like the multiplication of these than any morbid process with which he was acquainted. It was, however, quite possible that the structures might be found in other conditions, for he had not all kinds of pathological material at his disposal, and this was a part of the work which others might extend.

II. Original Communication.

CONTRIBUTION TO VENESECTION AS A REMEDY.

By John Shand, M.D., F.R.C.P. Ed.

Mr President and Gentlemen,—In response to the request of some of our ex-Presidents, I have selected the subject of Venesection. Though regarded as a *bête noire* or almost forbidden subject —indeed, a *questio vexata*—at various periods during the last three centuries, yet as a wave in its favour seems now impending, I feel it my duty to contribute a few cases from past practice in illustration of its utility.

I am the more encouraged to do this, as the example has been already set by professional men of such distinction as Dr Broadbent of St Mary's Hospital, London, the President of the last meeting of the British Medical Association in the capital of the United Kingdom; as also by His Excellency[1] the philanthropic Dr Gunning of Brazil, otherwise so well known in this city for his munificent contributions to our various schools of learning. Indeed, my cases which I propose bringing before you are to my mind much in the way of a complement to his own

[1] A title conferred on Dr Gunning by the late Emperor of Brazil for his benevolence and generosity to its inhabitants, on whose behalf he is even now *en route* to Brazil to institute an orphanage at his sole expense. By special permission of Her Majesty, Dr Gunning is permitted to use this title here as he did in Brazil, though it is a title usually limited to ambassadors.

paper on this subject,—which paper, on account of his impending blindness, I read for him, at his request, to this Society some six sessions ago. To my disappointment no discussion followed the reading of the paper—a striking proof how little palatable the subject is even yet, else what I have to say now would have been said then.

I may here be allowed to remark how much admiration I felt for the heroic moral courage of the man who did not hesitate to confess to a fatal error of practice during a period of ten years in Brazil, and who also felt no humiliation in adopting an entire change of practice in conformity with that of the local practitioners—in short, employing venesection, which he had discarded. This change improved his results to a proportionate extent.

The subject of venesection, if entered on historically, is of such magnitude, and is so amply discussed in our *Edinburgh Medical Journals* of 1856–7–8, that detail here on a topic already so exhaustively discussed by Alison, Christison, Alex. Wood, Andrew Wood, and Prof. Wm. Gairdner, on the one side, and Dr J. H. Bennett on the other, is quite unnecessary. Sir D. Maclagan referred his auditors of the Royal Medical Society to those journals, and I cannot do better than advise those who have not read them to do so at their convenience. The criticisms of Dr Gairdner, I may say, accord pretty nearly with my experience.

Prof. Sir D. Maclagan narrates his being bled at the age of 18 months. No higher testimony could be given to the harmlessness of venesection than his activity of mind and body at the present day, together with his distinguished career and the position he has achieved, both professionally and as a citizen of Edinburgh.

Frequency of Venesection.

Dr Broadbent writes:—"The spring and fall bleeding of our forefathers must have been a great relief to many of those upon whom it was practised, and in some, I have no doubt, it prolonged life. I have had under my care from time to time an old lady, the only clue to whose age is the fact that her father, who was a historical character, died in 1803. She had been bled fifty times, first for puerperal convulsions, and for their prevention afterwards. The punctures at the bend of the elbow had been so numerous that there was no room for more, and the later bleeding had been done from a vein in the foot. I do not think the old lady would have been alive now but for the bleeding. When I last saw her, about two years since, she was managing the family of a deceased daughter."

Although the habit of venesection was much modified in my time, chiefly from the influence of my predecessor, I knew and conversed with two men who were sure that considerably over

sixty times in their lives they had been bled,—for many years regularly at the rise and fall of the year. One of the two was generally bled on the roadside by one of the stonebreakers, an occupation that, in the South of Scotland, seems to have been combined with venesection, just as painter and glazier and tailor and clothier are.

I may premise that, like Dr Gunning, I commenced the first few years of practice without the use of the lancet, and was aided by the fact that for a time no acute cases presented themselves. In the Edinburgh Royal Infirmary, while I was a resident there, I had never seen a patient bled, and afterwards in the South of Scotland, I found what appeared to me a very decided change of type from acute to mild forms of congestion in various organs, but chiefly in the lungs. I attributed this change, which was so general, to a change in the mode of dieting, namely, from oatmeal porridge with good milk to tea and bread served often at every meal; and change of occupation, from outdoor to indoor work. Indeed, I found that little but dieting, poulticing, and rest was required.

As an apprentice to a doctor in my earlier days, I had imbibed what would now be regarded as a prejudice in favour of venesection. Afterwards when acting as a resident in the Royal Infirmary, and undertaking simultaneously the duty of assistant pathologist to Professor Hughes Bennett before he obtained his chair in the University, I abandoned my early views on venesection and largely adopted his.

This change was all the more easily effected, because, as above stated, I had never seen venesection performed on any of the patients under my charge, which may be partly accounted for by the fact that I cannot recall a single case of true sthenic pneumonia during my period of service.

I will here detail a case which chiefly effected my conversion again to venesection.

A stalwart septuagenarian, well known for his contributions to the history and local antiquities of the South of Scotland, consulted me for symptoms clearly indicating an impending attack of apoplexy. After asking his age, I remarked, "I daresay you wanted to be bled, but at your age a prescription will do better." Accordingly I prescribed a mixture no doubt containing tr. aconiti, etc. We parted, and I think three days afterwards I observed him in the street in front of me. I was struck by the comparative elasticity of his step. I hailed him. "How are you, Mr N.?" I asked. "Thank ye, Doctor," was the reply, "I'm *perfectly* weel." I observed a little hesitation in his manner, and an expression as if he feared to pain me. Then suddenly, as if he had made up his mind to a crisis, he resumed—"Weel, Doctor, I maun just be honest wi' ye. I didna like the look o' yon bottle at a', nor the paper neither, so I let them alane, and gaed to the

clogger" (a safe hand with the lancet, and a most intelligent man), "and had a wheen cups ta'en frae my airm." Then with a low, sweeping bow—"A' the same thanks tae ye, Doctor." He lived to be in his fifth year as an octogenarian, and died of hæmorrhage from internal organs. Would he have lived to a hundred if he had been bled, as in former days, twice a year? This has been a consideration with me, as it has been with Doctor Broadbent in his case mentioned above of the lady who was bled fifty times. My patient here mentioned was one of the two already referred to who had been bled over sixty times. Their practice was venesection twice a year, at the rise and fall, whether by doctor, minister, lady-practitioner, or stone-breaker.

Deeply interested in the history of my patient, I learned that his first venesection was in his second decade, in a case of strong fever. The arm broke out during the night, saturating the bedclothes. He awoke in discomfort, and summoned his mother, who in alarm sent off for the doctor (Nathan ——, if I remember the name aright). He lived a few miles off at a hamlet called Causewayend, now Castle-Douglas. "Did the doctor come?" I asked. "No; he just sent word, 'He'll be a' the better o't.'"

This apparently heartless response I interpret as a proof of the sagacity of the doctor, who I suspect had on reflection thought he had not bled enough, considering the violence of the attack and the powerful frame of the youth.

In a village hard by there was a famous Jane Sproat who practised the art, and the Lady Grierson of Rockhall had a very high repute in the first quarter of the century. I was told that "folk went from all walkable distances" to be bled by her ladyship, whose social position lent an additional charm to the performance. Indeed, the clergyman, the dominie, and the smith, with a few amateurs, all practised this art. In proof of the clerical performance you will find a record of it in Dr Russell's book on *Yarrow*, where he says that on the Monday after the Communion all who wished to be bled were requested to go into the vestry. In the beginning of this century and the end of last, no young man belonging to a family of any position ventured on the Continental tour without being thoroughly coached in the art.[1]

Personally, I may say, that a courteous offer of venesection was once made me by a gentleman of the old school, an octogenarian, who addressed me after my horse had fallen with me in descending a steep hill. He begged me to step into a cottage close by and he would bleed me. I declined with thanks, saying that my hat had saved me. He repeated his offer, and said he

[1] *Vide* Waterton's *Wanderings in South America*, where he bled himself for an attack of fever, and with perfect success. And again, at his own estate in Yorkshire, fearing mischief from the blow of a branch, which fell upon the back of his head, he used venesection with equally good result.

always carried a lancet somewhere in the recesses of his habiliments for the benefit of himself or friends.

Cases.

I will now briefly describe a few cases bearing on venesection.

Pneumonia has been the field in which the battle has been principally fought, and coincidently it happens that Dr Gunning's cases are likewise pneumonic.

According to a letter in the *Lancet* a year ago the stages of pneumonia are still far from being definitely defined, even in our text-books. What is known as congestion has such a width and uncertainty of meaning that from the simplest catarrh up to almost engorgement, with febrile pulse and quick respiration, all pass currently under the same denomination.[1]

The cutting short of Pneumonia by Venesection.

Case I.—Some twenty-five years ago, my then assistant, the late Dr Cumming of Wigtown, was attending a case of pleuro-pneumonia some four miles off, and his daily report becoming less and less satisfactory, and no venesection having been used, I on the fourth day arranged to visit the patient with him, and found the case so serious that I sat cogitating at the bedside several minutes before I decided on bleeding him at the arm. Knowing that bleeding on the fourth day was exceptional, I used my lancet with my left hand, and kept my right ready with a pledget of lint to arrest the bleeding the moment I perceived it was not answering the wished-for object; but I had hardly begun when a gradual improvement in the pain and dyspnœa ensued, and I continued the venesection till I considered the amount sufficient to produce a favourable turn in the disease. Next day the report was brought me by the cotman's master, an intelligent farmer, reliable for counting the pulse and respiration correctly. After I had seen him Dr Cumming came to me inquiring what the report was. "What was the pulse?" he said. "80," I replied. With a look of cynical incredulity at what he considered the absurdity of the report, and turning on his heel, he responded, "180, more likely." However, the daily report rendered any further visits unnecessary. To use the South country expression, the man "never looked over his shoulder again."

Case II.—At a time of sickness in my family which debarred me

[1] In cases of true first stage of pneumonia, I have regarded a modification of the normal sounds (as if heard through a coating of blanket), with a corresponding dulness on percussion, as sufficient evidence in the physical symptoms of the chest to warrant venesection. The degree of fever, pain, thirst, and especially of dyspnœa, should help in deciding on the quantity of blood to be withdrawn. Crepitus is a stage beyond this, and proportionally less favourable for having the attack arrested.

from practice, and when I had Dr F. R. Cadell as my *locum tenens*, and Dr Harry Hay, now of Leslie, Fifeshire, as my assistant (both of whom, I need scarcely say, had been educated in the Dr Bennett theory of no venesection in pneumonia), I was asked by Dr Cadell to see a man in the surgery, who, he thought, I might consider should be bled. On seeing the man, a rapid and cursory examination with ear and fingers satisfied me of congestion existing in both lungs, with all the symptoms of going on to the stage of inflammation or pneumonia. Accordingly I bled him on the spot, and as he was a man of sthenic habit, I literally followed out the well-known Cullen's prescription of a pill of calomel and opium—one immediately after the venesection, and one, minus calomel, at bedtime for three nights.

I heard no more of the case at the time, but some eighteen months afterwards a woman presented herself, who turned out to be the man's wife, and said she called to thank me before leaving the district for another dairy elsewhere, informing me at the same time that her husband had walked home after the venesection—four miles uphill—and was at work in three days or four, and had remained quite well ever since.[1] Was this amelioration of symptoms, or cutting short the disease?

Case III.—Another cotman, some three miles off, was put under my care after an attack of pneumonia, for which he had not been bled. I found him with his life hanging by a thread. He had been kept alive for several weeks by the kindness of neighbours, who had supplied him with soups and jellies. He made a slow and precarious recovery. Knowing the likelihood of a return of his attack in the hay season, on bidding him good-bye I told him, if he should be attacked again by the same symptoms as at the beginning of this attack, he was at once to get himself bled, if not by a doctor, by any one who could bleed him—not difficult to find then, at the same time giving him Cullen's prescription to follow. Some ten or twelve months afterwards, as I had started in my saddle, I was hailed by a man who blocked the way, bawling out, "I hae lost the line!"

On recognising him as my old patient, I pulled up and said, "Are you ill again?" "Yes, and I've been to the clogger (reliable bleeder) and been bled, but I hae lost the line." (This was Cullen's calomel and opium pill.) I turned my horse, telling the man to follow, and at the chemist's gave the required prescription.

Three, or at the most four days afterwards, I visited the cottage where I had attended this patient, but found he had removed three miles further down the coast. I followed up, missed him, but saw his wife, who said, "He's doon at the hay, and quite weel."

[1] Dr Cadell lately told me that No. II. Case was the first case of venesection he had ever seen, and the last. In neither of the above cases had crepitus been reached.

I know I should have heard had anything gone wrong, but I never did hear. I repeat the question, Was this cure, or amelioration only?

Congestion, or subacute first Stage.

Slight.—Four or five cases occurred of sickness sufficient to confine the patients to their beds, without any urgent symptoms, but sufficient to need the doctor's advice—so the friends thought. In all these cases the symptom most complained of was *constipation*, although in each I found slight dyspnœa, and very slight occasional cough and absence of appetite. An examination of the chest revealed in each case subacute congestion of one or both lungs, but only in patches. Rest in bed, with poulticing, and a few leeches at the hollow above the sternum in two of the cases, which were more decided than the others, with a mild opiate and mild aperient, was all that was needed to set matters right. There was no epidemic in the district to account for this.

I must remark, also, that in the two cases where leeches were applied, followed by a gentle opiate, the constipation yielded before any aperient was given. Probably the metastasis of blood from the vessels of the abdominal viscera to those of the chest might account for the constipation. At all events, the next case remarkably illustrates this view.

Case IV. (Apoplexy of Lung).—Called to see a young man of about 22, of remarkably good physique, whose friends told me that constipation was all they thought the matter with him, and that it had resisted the ordinary aperients employed in domestic medicine. On my way to visit this patient, the question occurred to me, Can this be another case of congestion of the lungs? I found it to be so, and of both lungs, to an extent that I might call engorgement or apoplexy. The propriety of venesection was uppermost in my mind, but, understanding that the patient's father was a medical man in large practice in England, and being unable to ascertain his views, and immediate action was necessary, the question was simplified by my being recalled suddenly to the patient's room. I found Nature was undertaking the doctoring herself, and that blood was gushing from both nostrils and mouth in an impetuous stream. His brother-in-law, whom I had taken into my confidence as to venesection, quietly observed to me, "You will not bleed him now?" "I don't know," I responded. Afraid that the loss of blood might be greater than was useful, I opened a vein widely, and before three ounces had escaped the hæmorrhage at mouth and nostrils at once ceased, as I had hoped, and I continued the bleeding at the arm till I thought a satisfactory impression had been made.

The case was accompanied by all the symptoms of inflammation

of the lungs, regarding this as the congestive stage. Of course, Cullen's opiate was administered, and the improvement next day was so great that the remaining treatment consisted chiefly of the neutral salts, especially the citrate of potass in an effervescent form. His father had arrived by this time, and his views proving to be uniform with my own as to venesection, he congratulated me on having saved his son. In his own practice, he said, he had lots of pneumonic cases, but, with few exceptions, of the mild subacute form, not demanding venesection. The patient made a rapid and satisfactory recovery, the venesection or the opiate, or both, removed the constipation, as in the preceding cases. Possibly Nature's loss of blood might have answered, but I felt more comfortable in treating by venesection.

Apoplexy (One Case).

Case V.—Sent by my senior to visit a lady attacked by an illness partaking of apoplexy, so termed, as this was her third attack in three years. I was starting without my lancet, but happened to remark to my senior, "Should I take a lancet?" I saw the response on his face, and immediately put myself in possession of the little weapon. During my ride I reflected as to the propriety of using or not using it. On arriving I found the question settled, not this time by Nature, but by accident. The lady had fallen against the sharp corner of a table and wounded the temporal vein, and was able to welcome me on entering the house. She conducted me to the scene of the accident, where I found a large coagulum of venous blood, about 4 inches deep vertically, and from 4 to 5 across the base. So far the treatment had been very successful, and I did not find it necessary to do more than enjoin rest in bed and arrange the diet. The lady died several years after, but during the interval had a longer immunity than usual from any attack.

Pain (Two Cases).

I will now give two cases in illustration of the complete relief to excruciating pain. These both relate to the eye.

Case VI.—Called to a case late of an evening, but was unable to attend to it till next forenoon. A stalwart young farmer had got his eye hurt by a branch, if I remember aright. When the doctor did not appear, the mother, in her anxiety, had some conversation with the smith hard by, in which he said that "if it had been a horse he knew what he would have done." "What would you have done?" "Bled it," he replied. "Well," rejoined the mother, "do you think you could bleed my son?" "Oh, aye; I *have* bled, and I wouldn't be afraid to bleed again."

The son *was* bled—to the usual extent, no doubt, of three or four cups. When I arrived next forenoon, with smiling faces they informed me there was no patient—the smith had cured him. And so it was. I am glad to say no bad consequences followed. The gentleman is well to this day.

Case VII.—Corresponding to this case I will shortly detail another, not dissimilar, but unfortunately much more severe. Being a guest at the house of one of our merchant-princes in the neighbourhood of Manchester, an accident occurred as the gentlemen of our party were playing a game at billiards. I was lifting my cue at one end of the table to play, when a noise at the other end, as of some one stumbling, attracted my attention, and looking up I observed one of the party recovering from a stumble, with one hand covering his eye, while the blood gushed from between his fingers. He had fallen on the broken end of a cue. Instantly I rushed to rescue him. One look at the eye exhibited the pupil dilated to the utmost, and by the touch I perceived that the eye was becoming hard as a stone. Not to add to the alarm, as the ladies of the party were all seated round the room, I conducted my patient to an adjoining apartment, and got what fortunately was in the house, viz., ice, which was kept constantly applied, till the medical attendant of the family and the oculist, Mr Windsor, should arrive. The pain was to some extent ameliorated by these applications, but at times was almost unbearable. I was invited in to a consultation with the two gentlemen, who very quickly arrived; and on being asked to express an opinion, I narrated to them the case I have just detailed to you, and it was agreed to perform venesection. Suffice it to say, although I had to supply the lancet from my dressing-case, the operation was completed by the medical man, who said he had bled once or twice in his younger days. The patient was a splendid specimen of physique, and I rather think that Cullen, or even Alison, would have withdrawn more blood or repeated the venesection after a few hours.

Although the oculist hoped the sight might be restored, it was not. The only comfort—and that not a small one—was that next day the patient said—"Gentlemen, I must tell you that by this time I must have been either dead or in an asylum from the pain I was suffering, had it not been entirely and immediately relieved by the opening of the vein in my arm." There was no return of pain or inflammation, but the sight was gone.

This case appears to me precisely what Dr Alison describes—without venesection it must have gone on to suppuration and sloughing of the eyeball.

In regard to Case IV. I may here quote to you the interesting case of the late Earl of Malmesbury, which wonderfully

corresponds, as a traumatic or surgical case, to the above idiopathic or medical one of the gushing of blood from the mouth and nostrils.

In Lord Malmesbury's *Life* it is stated that at the age of 21 he visited Geneva, and with a friend went to take lessons in fencing from the then celebrated teacher who lived there. While engaged with their foils, the button of the fencing-master's foil had come off, and the sharp-pointed weapon passed through his Lordship's right lung. He, fancying he had received a blow on the shoulder from the friend who accompanied him, turned round to remonstrate. As he did so the blood gushed from his mouth and nostrils. All the professional skill of Geneva considered the case desperate, but a surgeon of the old Napoleon (Le Grand) happened to turn up. He regarded the case hopefully, and quietly said in French, "Why, he's only 21; bleed him till he faints." This was accordingly done, and he made a tolerably rapid and steady recovery. He died only two years ago, aged 84 or 85.

This case bears a striking parallel to Case IV., where the gushing of blood arose from congestion of lungs. In both the treatment was the same, and in both proved successful.

Stenosis of Mitral Valve.

Dr Broadbent in 1887 wrote strongly on the curative effect of venesection on this state of the system, detailing particulars of one case.

Case VIII.—Some years ago I was called to such a case—a young lady of 17. Pain and dyspnœa were urgent. At first sight it simulated an attack of angina pectoris. The capsules of nitrite of amyl were then unknown, and after some hesitation I opened a vein of the left arm with my left hand, noting anxiously the effect of each drop, I might say figuratively. She got perfect relief by the time I had taken a wine-glassful, say 2½ ozs. Again I was called in the day after, and repeated the operation to the same extent, with similar perfect relief. A third time I was summoned a few days after, and again opened a vein. This time the relief proved permanent, and I had the gratification last year of seeing my quondam, graceful, slim-figured young patient, now a becomingly "fat, fair, and forty" middle-aged lady. She told me she had had an immunity from serious sickness from that time, but said I bled her only twice, explaining that the second venesection was so soon after the first that I did not use my lancet, but only sprung the vein with the bandage, which confirms the statement that the interval between No. 1 and No. 2 must have been only one day.

Case IX.—Although I succeeded in the above case, I lost my next by procrastination. I used stimulant and sedative treatment,

with partial relief; but general dropsy followed in a week, and I lost my patient. This case was congenital. It is on my conscience to this day.

I can scarcely conclude this paper without admitting that on one occasion in '57 my curiosity and interest upon this vexed question of venesection was so overpowering, that I retired to my room and bled myself to the extent of between 30 and 40 ozs. into a washhand basin. The marks are still visible. So little inconvenience did I experience that I drove in the evening some six miles to dinner, and, though I was told I looked pallid for some days, I cannot recall that I was in the least inconvenienced by it.[1]

Indeed, gentlemen, I entertain a high opinion of the utility of venesection when performed in cases suitable for it, and I am quite willing, like Dr Gairdner, to allow any theory, provided it does not interfere with my practice.

In addition, practically speaking, I would not hesitate to employ venesection in a case of whatever nature that exhibited asthenic circulation, endangering any of the vital organs.

In illustration, I recall a case of a gentleman of about 30 years of age, of excellent physique, of the nature of whose case on my first visit I was unable to form a definite opinion. There was no epidemic prevailing at the time, but on my second visit my apprehensions were increased, in case of an attack of cerebral or pulmonary apoplexy, from the character of the pulse and the febrile symptoms. In short, I opened a vein, and took a fair bleeding. Satisfied that I had probably saved him from an immediate vital danger, the day following I found he had passed a much easier interval, and that a case of modified small-pox was now distinctly expressed. The case ran a benign course.

A case analogous to this in London came to my knowledge afterwards, where a lady lost her husband by his being overtaken with acute apoplexy before the small-pox had expressed itself.

In this case there was an epidemic of small-pox in the district, and the patient had never been vaccinated. In the previous case there was not another instance of small-pox in the district.

I have confined myself to venesection in respect of blood-letting, though I have also been much indebted to the use of local depletion, chiefly by leeching, in elderly people.

From a letter I received from Dr Gunning a few months ago I extract the following sad passage:—

"The attempt to read my paper, and afterwards to revise it for printing, was the last blow to my sight. I never recovered from the nervous depression and irritation thus caused; and no skill which I consulted could put right the obstinate glaucoma which ensued." Near the end of his letter he says:—"I should not like to be the man to let a patient with pneumonia, acute bronchitis,

[1] I remember that not many days after my venesection I overtook some of the hardest physical work in saddle I ever had.

or pleuritis, go on without the use of blood-letting. It would be ever on my conscience."

I have many historical data of the good effects of venesection, but I must close with a quotation from Virgil, which I had intended to preface my paper with.[1]

> "Quin etiam, ima dolor balantum lapsus ad ossa
> Quum furit, atque artus depascitur arida febris
> Profuit incensos æstus avertere et inter
> Ima ferire pedis salientem sanguine venam."
>
> —Virgil, *Georg.* iii. 457.

> "Deep in their bones when fevers fix their seat,
> And rack their limbs, and lick the vital heat,
> The ready cure to cool the raging pain
> Is, underneath the foot to breathe a vein."
>
> —Dryden.

This very day I met an elderly man, who told me his mother is still alive, aged 90, and that in early life she had been well coached in venesection by the grandfather of a distinguished member of our Edinburgh staff, and exercised the art so lately as a year ago, and felt convinced on the authority of medical men in one of our northern counties that she had saved many lives. My informant's grandfather lived to the age of 101, and had undergone venesection annually for many years of his life. I have reason to believe the information is reliable.

After all I have said, still great prudence must be exercised in the selection of cases suitable for venesection, and as to the quantity of blood to be withdrawn.

Addenda.

Professor Alison and Dr Pherson.—"He's a wonderful man, that Alison," said Dr Pherson, of Cape Town to me one day in my studentship.[2] "You know I'm attending a case in Nicolson Street, and finding it was apparently sinking, I caught Alison at his class door, and, though I thought the woman had no spare blood left in her, he opened a vein yesterday, and she is one-half better to-day already."

Charles II. was saved by a chance doctor who bled him when he fell in a fit of apoplexy in his palace (see Chambers' *Book of Days*).

Wounded Soldier.—On one of the battlefields of the Peninsula an Irishman supposed to have been mortally wounded was found

[1] Though Latin is more than ever a dead language with the majority, yet some of my auditors may find for themselves how different it used to be, and possibly find on the shelves of the College of Physicians a Latin Thesis of a forefather, as I did that of my own father after I became a Fellow.

[2] Dr Pherson was a graduate of ten or twelve years' standing.

to be still living. "Och," said he, "I was kilt, but a doctor came and bled me, and I'm all alive again."

Conclusion.—Like Dr Gunning, I am not particular about theories, and like him, I can adopt the exudation one in conjunction with venesection.

Professor Gairdner likewise says he is "willing to make a bonfire of all theories past, present (and probably future), provided the practice is correct."

I hope the Society will pardon any prolixity I may be accused of on this subject, but I feel that the time has come when venesection is recovering from the disrepute into which it had fallen, though I do not conceal from myself the danger, if the tide against venesection has indeed turned, of its possibly being carried to an extreme, as has happened more than once before.

Meeting IV.—January 21, 1891.

Professor A. R. Simpson, *President, in the Chair.*

I. Election of Ordinary Members.

The following gentlemen were elected Ordinary Members of the Society:—Dr Ralph Stockman, Hope Street; and A. Lockhart Gillespie, M.B., C.M., 10 Walker Street.

II. Exhibition of Specimens.

1. *Dr John Playfair* showed the KIDNEYS, URETER, and BLADDER from a case of hydronephrosis. The patient from whom this specimen was obtained was a male infant aged six months. When a month old, a swelling was noticed on the left side of his abdomen. On admission to Hospital, on 1st October 1890, he was thin and weak-looking, and weighed only $8\frac{3}{4}$ lbs. All his organs gave normal physical signs. He was stated to micturate without difficulty, and his urine was found on examination to have a sp. gr. of 1019, and to be free from albumen and sugar. His abdomen was somewhat swollen, and on palpating it, two rounded, rather tense masses were made out. One mass felt about the size of an orange, and occupied the right lumbar region; the other was much larger, and felt oblong in shape, and filled the left lumbar and iliac regions, and passing downwards below the level of the umbilicus, it reached the right iliac fossa. No fluctuation could be detected in the swellings. The abdomen continued to increase in size, and a week or two after admission, as the diagnosis was still doubtful,

patient was put under chloroform, and a more thorough examination made. Three distinct masses could now be made out in the abdomen —one in either flank, and a third in the middle line below the level of the umbilicus. The swellings felt softer than formerly, and though no fluctuation could yet be detected, they gave an impression of containing fluid. A hypodermic syringe was, accordingly, passed first into the central mass, and then into the other two, with the result that a clear fluid was readily obtained from all three. On testing the fluids, each was found to give an acid reaction and to be free from albumen. Microscopically the fluids were found to contain a few nucleated epithelial cells and leucocytes. The case was then diagnosed as an hydronephrosis. Patient gradually became weaker, and died about a month after admission. At the post-mortem the pelvis and ureter of the right kidney were found greatly distended. The ureter was very tortuous, and dilated in its whole length to about the size of the child's small intestine. The right supra-renal body was normal in position and size. The swelling on the left side of the abdomen was found to be composed of œdematous connective tissue overlying an enlarged and cystic kidney, which was almost completely collapsed. The left ureter was dilated, but only to about half the size of the right. The bladder wall was much thickened, measuring about a quarter of an inch. The vessels on the anterior surface of the bladder, over an area about the size of a penny, showed small varicose dilatations. A No. 2 catheter could be easily passed along the urethra into the bladder, though the prepuce was absolutely adherent to the glans. The adhesions could be torn by the finger-nail, and the prepuce retracted. I think the most probable explanation of the state of the parts was that suggested by Dr James when the specimen was exhibited. He thought that the adherent prepuce had set up a condition of irritable bladder, which had thrown that organ into a condition of almost constant contraction. This would lead to its diminution in size and hypertrophy, and would also close the openings of the ureters into the bladder, and so cause dilatation of ureters and kidneys.

2. *Dr Alexis Thomson* showed a LARGE FIBROMA OF THE DURA MATER, occupying the right half of the posterior fossa of the skull, and causing almost complete atrophy of the lateral lobe of the cerebellum on the same side. The tumour was egg shaped, measured 5·5 cm. and 3·5 cm. in its long and short diameters respectively, and was attached by a narrow base to the dura at the point of junction of the lateral and petrosal sinuses. Its surface was invested by a thin fibrous capsule, and showed several projecting nodular elevations, one of which projected through the tentorium, and appeared as a small pedunculated mass on the superior aspect of the latter. The substance of the tumour was firm and fibrous, pinkish-white in colour, and the cut section presented the watered-silk appearance. Its microscopical structure was that of a simple

fibroma. The specimen, along with the base of the skull, was removed post-mortem from a man, aged 66, who died in the Norfolk County Asylum. A maternal uncle of the patient had died insane. The patient himself had met with a severe injury to the head twenty-five years before his death; he was thrown out of a public-house, and alighted on the vertex, after which he was unconscious for ten days. On recovering from this state he became irritable, quarrelsome, aggressive, and addicted to fire-raising. On being convicted of arson and committed to prison he was found to be insane, and was sent to Broadmoor, and finally to the Norfolk County Asylum, where he remained till his death. He was described as a square, thick-set man, very feeble on his legs, able to move about with a slow, shuffling gait. He always complained of constant pain at the vertex and of noises in the head; he was never sick; his imperfect vision was attributed to presbyopia. Mentally he was irritable, melancholy, dull, did not employ himself, was cleanly, rational in many ways, artful and cunning, and had occasional outbursts of maniacal excitement. He remained like this for many years, and died from ulcerative colitis and diarrhœa.

3. *Dr Caird* showed for Mr E. Neve, Cashmere, a URETHRAL CALCULUS, removed by incision of the urethra from a male patient, aged 25. A second stone was pushed back into the bladder and dealt with by litholapaxy. The patient made a good recovery.

III. Original Communications.

1. RECENT WORK IN ABDOMINAL SURGERY.

By J. Halliday Croom, M.D., F.R.C.P. Ed., F.R.S.E., Physician to the Royal Maternity Hospital; Physician to, and Clinical Lecturer on Diseases of Women, Royal Infirmary; Lecturer on Midwifery and Diseases of Women, School of Medicine, Edinburgh.

Since my last communication to this Society I have completed a further series of 60 cases of abdominal section, and as some of them presented features of special interest, I have ventured to record them shortly.

1st, A group of cases of ovariotomy complicated with twisting of the pedicle. I have met with 4 such cases in my last group of 60, and altogether, in 260 operations which completes my experience, the condition has occurred six times. This accident is at once a comparatively frequent and interesting one. It is, further, a very serious one, as records of the post-mortem room abundantly show.[1]

Perhaps the most complete essay on the subject is that of

[1] Rokitansky described 13 cases, of which 8 were found in the post-mortem room.

Knowsley Thornton,[1] who found 57 cases out of 600 upon which he had operated. Schroeder out of 94 cases of ovariotomy had 13 cases of twisted pedicle. Nothing supports the plea for early ovariotomy more than these cases. Nothing indicates more clearly that an ovarian tumour should be dealt with as soon as it is diagnosed. Take three illustrations, one of delayed and two of prompt interference.

Case 259.—Mrs T. was seen by Dr David Menzies on Thursday 25th Dec., in acute suffering and somewhat collapsed. The pain came on suddenly after some slight exertion, and was localized to the lower abdomen, accompanied by vomiting. When seen later on, the abdominal pain was general and intensely severe, and could only be modified, not entirely relieved, by large opiates. Through the tender abdominal walls a tumour could be recognised, cystic in character, and passing up from the pelvis on the right side to midway between pubes and umbilicus. The nature of the case was obvious enough. Two days after, the tumour was removed with entirely satisfactory results. The temperature at the time of operation was 103°, and pulse 130. The relief to the patient was immediate. The tumour showed intense congestion, not only in the walls and contents of the tumour, but in the tube also. The pedicle was twisted four times.

Case 234.—Was seen by Dr Cappie on the 16th September, apparently suffering from acute peritonitis, which had come on early in the morning after an extra exertion in cleaning. Dr Cappie recognised the general peritonitis, and passing his hand over the abdomen felt the tumour. Next day he asked me to see the case with him.

I operated two days after, while still the peritonitis was marked, and removed the tumour, which is represented in the accompanying figure (Fig. 1), showing a well-marked twisted pedicle (there were altogether twelve twists), and though the adhesions were many, they were recent and easily managed. The patient did absolutely well without a single hitch.

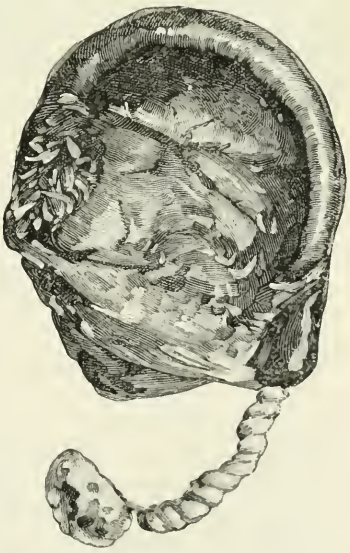


Fig. 1.—Ovarian Tumour. Twisted Pedicle: Twelve Twists.

[1] Thornton, *International Journal of Medical Science.*

Compare this, again, with case 210, which had been seen early in November 1889, and diagnosed as an ovarian tumour, but the recommendation of the physician who saw it was that nothing should be done beyond the application of a bandage. This was done. What occurred to her in the meantime I do not know, beyond that she pursued her ordinary avocations. In any case I saw her six months afterwards with acute peritonitis. The ovarian tumour was there beyond a doubt. I operated some days afterwards, and found the tumour dark, black, and almost gangrenous, surrounded by numerous adhesions, peritoneal, intestinal, and, indeed, general. These were managed in the usual way, and the tumour removed. The patient died very shortly afterwards, and no wonder. If she had been operated on six months previously, it is as absolutely certain as anything can be that she would have been alive now. The following figure (Fig. 2) shows the extreme attenuation of the pedicle, not being larger than a crow-quill.

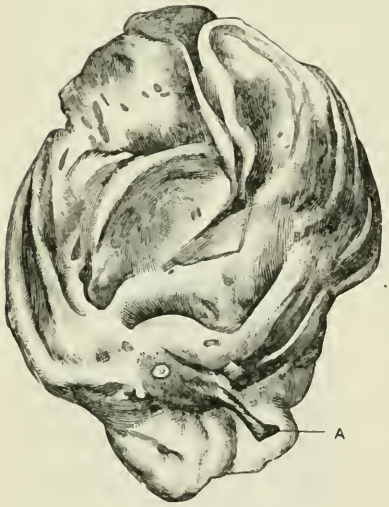


Fig. 2.—A, Twisted Pedicle, size of crow-quill.

I am well aware that Knowsley Thornton denies that gangrene ever occurs as the result of twisting of the pedicle, but I mean to say that the tumour I now refer to was as distinctly cut off from all circulatory help through the pedicle as any tumour could be. It will further show the nature of the tumour when I say that the adhesions were old and firm, and by no means vascular. It will not be difficult to understand that the tumour was not very different from a gangrenous mass.

The sort of growth that Thornton refers to is more a matter of transplantation, and occurs in those tumours which are dermoid, and which are nourished even after strangulation of the pedicle by their vascular adhesions.

These three cases illustrate the condition very fairly. In all of them the adhesions were extensive, and to these, along with the peritonitis, the danger of the case is mainly due. The contents in each case were clotted and dark fluid blood. I did not meet with any one containing pus, nor did I meet with a dermoid tumour, which Thornton says is so frequent under such circumstances. The diagnoses were not difficult: the presence of the tumour and the sudden and acute pain over the region of it, with more or less collapse, were sufficiently indicative of the condition. Of course, acute peritonitis very often masks the condition, but a

careful examination into the history and local conditions will, in most cases, make the diagnosis clear.

It seems very difficult to account for the accident. Tait attributes its occurrence to the passage of fæces down the rectum. This may be the case in many, but obviously cannot be in all, because the twist is by no means always in the same direction, viz., from left to right, but frequently from right to left.

Thornton suggests that in the cases in which he met with it the patients were multiparous and young, and in active menstrual life, and the twist was the result of some sudden accident or strain. He also refers to tapping the cyst, and leaving it as a possible cause of twisting, and I can readily understand how this takes place, though I have not met with cases of the kind.

Alban Doran explains it by the presence of the costal cartilages above and the pelvic structures below, the tumour moving freely laterally and anteriorly.

In two of my cases the accident seemed to occur after the tumour had just emerged from the pelvis. Being freed from the restraint of the pelvic walls, the cyst enlarges rapidly, and being movable in the abdomen, the pedicle becomes stretched and the tumour tense, so that the movement of the intestines and abdominal walls causes it to rotate on its own axis. It is only by some such recurring cause such as this that ten or twelve twists can be accounted for. Any accident or strain may cause one, but the difficulty is to explain the repeated twist. In the two cases I refer to, the tumours were known to exist in the pelvis previous to the occurrence of the twist, and in both cases the patients were conscious of rapid abdominal enlargement. It seems likely that the filling and emptying of the bladder, giving rise to increased or diminished abdominal pressure, may play a more active part in the production of these repeated twists than the rectum.

The next case of interest is one of cholecystotomy. The patient, a married woman of 45 years of age, was sent to me by Dr Macleod of Hawick as suffering from gall-stones. She was suffering pain over her liver, and had repeated attacks of jaundice. I could feel no swelling over the border of the liver, only an obscure tenderness. As her attacks of jaundice were frequent, however, and the tenderness over the liver, though slight, yet continued well marked, I arrived at the same conclusion as Dr Macleod, that she was suffering from gall-stone.

The ordinary incision was therefore made in the side, and with some difficulty the median fissure of the liver was reached, and the small shrunken gall-bladder with difficulty recognised. It was not bigger than a filbert, and was firmly contracted round the gall-stones. It was drawn with difficulty to the surface and incised, and two gall-stones were extracted. The gall-bladder was sewn to the abdominal wall, and the fistula treated in the ordinary way. The operation was a long, tedious, and difficult one, lasting over an

hour and a half. The patient was well that night and the following day, but died suddenly with obscure symptoms, and as no post-mortem was allowed, I cannot give any definite explanation of the untoward result.

The extreme contraction of the gall-bladder was interesting, and it is entirely due to the information gained from Mr John Duncan that I was able to recognise the gall-bladder in its small and attenuated state, he having described a similar condition. When the gall-bladder is large, and forms a distinct cystic tumour, it is sometimes apt to be mistaken for a kidney; but when the distension is moderate, the diagnosis of the condition is simple, and the operation comparatively easy. This extreme contraction of the gall-bladder rendered it very difficult to bring it to the surface, and still more difficult to stitch it to the walls.

Küster's method seems to me in such cases more satisfactory and much easier, namely, to extract the calculi and close the internal wound. This operation has been performed repeatedly and with satisfactory results, notably by Küster,[1] Zielewicz,[2] and Stewart.[3] Another, easier still, would be the operation of cholecystectomy; but then it has not yet been shown that the gall-bladder is superfluous, though two cases have been recorded where the operation was immediately successful, though what was the ultimate effect I am unable to say.

Though the issue of my case was unfortunate, yet the general result in gall-bladder surgery is entirely satisfactory. For instance, Mayo Robson had 14 cases, all of which recovered, not to mention Tait's brilliant results in this form of interference. Kocher, Sanger, Thornton, and many others, all record lists without a single death. Of course there will always be two great sources of danger both in cholecystotomy and cystectomy, viz., bleeding and peritonitis, for one can never be sure that the stitches in the gall-bladder will hold, for the walls themselves are diseased.

These operations are not by any means easy when the bladder is small, as my own case indicates; but statistics show that, with due care, they are attended with little danger, and what risks do exist are fully compensated by the great relief obtained. Of course the diagnosis of gall-stones will always be difficult, and twice I have refused to operate because I could not satisfy myself of their presence. Certainly the symptoms are very uncertain, and, unless the tumour can be felt, the diagnosis is doubtful.

I have been obliged to resort to the enucleation of sessile tumours on three occasions.

These tumours have invariably originated in the broad ligament. They offer, perhaps, the greatest difficulty of any set of cases that

[1] Dr E. Küster, *Archiv für Klinische Chirurgie*, vol. xxxvi., 1887.
[2] Zielewicz, *Centralblatt für Chirurgie*, May 31, 1888.
[3] Dr R. N. Stewart, *New York Medical Journal*, May 25, 1889.

I have met with. When such a case occurs, after the hand has been carefully passed round the whole site and the relation of the tumour carefully settled, one of two things can be done. Make an incision in the peritoneum, say, in the middle of the tumour, and set to work to peel the peritoneum straight off it, securing any vessels with forceps as the case proceeds. Ultimately, when the base of the tumour is reached, it is simply lifted out, and the bare bleeding peritoneum left beneath. The peritoneum is then ligatured by an ordinary knot at the base, and the redundant peritoneum removed. This, however, is not uniformly possible. Sometimes, as in one of my cases, the peritoneal base was so big and broad that it required to be stitched by a whole series of ligatures.

In the one case where I was obliged to do this the patient made an excellent recovery; and in one other case, where the peritoneum was easily stripped off, easily ligatured, and equally easily removed, the patient died, the reason being that in the former case the tumour was small and the shock comparatively slight, whereas in the other case the tumour was half as big again as an ordinary full-time pregnancy, and had been repeatedly tapped before I saw her, and the shock on removing the fluid and stripping off a large amount of peritoneum was, of course, correspondingly great.

Certainly enucleation of ovarian or broad ligament cysts is an enormous addition to the possibilities of abdominal surgery, and though requiring care and trouble, gives, at least when the tumour is small, excellent results.

The following case illustrates the need of careful drainage and washing:—

The most interesting of the three was case 201. She was a lady of 38 years of age, sent to me by Dr Macnee of Inverness, and on opening her abdomen I found an ovarian tumour about the size of an ordinary football. After emptying the cyst and separating the adhesions above, I found there was no trace of pedicle whatever. I therefore proceeded to enucleate, and having made an incision in the peritoneum towards the base of the tumour, I succeeded in separating the one from the other. The remaining peritoneum and broad ligament had to be secured by separate ligatures in three places. The peritoneal cavity was carefully washed out, but no drainage-tube was used. Three days afterwards patient showed symptoms of internal hæmorrhage; I therefore placed her under chloroform, and re-opened her abdomen and removed several pints of dark fœtid blood. I again washed her abdomen carefully out, put in a drainage-tube, and for six weeks there was a continuous outpouring of dark fœtid blood. Each day the cavity was washed out; and at the end of two months she went back to Inverness quite well.

The lesson I learned was the value of the drainage-tube from the first. This I remedied in my other two enucleations. I feel

confident that the drainage-tube is essential to success in these cases.

In my last sixty cases of abdominal section there have been three hysterectomies, all of which proved successful. The first two were for fibro-cystic tumours of the uterus. The only points of interest about them were that in both I freed the broad ligaments; and what seems to me equally important, in both I used the elastic ligatures, through which I put ordinary pins, and then allowed the pedicle to slough off. My previous experience of all forms of metallic clamp had been so unsatisfactory, that, indeed, until the three to which I now refer I had never had a successful hysterectomy. I mean, of course, by abdominal incision. To the last of the hysterectomies I wish to draw special attention.

I. H., aged 25, a dwarf, was recommended to me by Dr Basil Orr as a suitable case for Cæsarean section in February 1890. She gave the following history:—She had noticed a swelling in her abdomen for the last four months, which was rapidly increasing in size. She had not menstruated since September, seven months ago, but otherwise was in good health.

On examination she presented all the usual signs of pregnancy, the uterus being, however, drawn up entirely out of the pelvis, which gave the following measurements:—

Conjugata vera,	1½ in.	Interspinous, . .	8½ in.
Conjugata externa,	5¼ „	Intertrochanteric, .	10¼ „
Intercristal, .	8¾ „	(Height of patient, 3 ft. 8	„)

Of course, delivery *per vias naturales* was an absolute impossibility; therefore operation was recommended about the middle of ninth month; but as some uneasiness and suspicious labour symptoms showed themselves at the end of eighth month, it was thought advisable to perform operation at that time.

The question then arose as to the nature of operation to be undertaken, and the choice lay between Cæsarean section and hysterectomy by Porro's method. Cæsarean section has given excellent results in Germany, and the remarkable series of successful cases of Dr Cameron of Glasgow entirely bear these out. In my case the special indications for a Porro-Cæsarean section were not present, viz.—

1. When the uterus is the seat of any disease, such as myoma, which if left behind might soon require removal.

2. When the patient has already been so long in labour that it is probable that the discharge is beginning to decompose, or when she has been examined by persons who may easily have infected her, or where she shows evident signs of infection having taken place.

Although this girl presented none of the indications insisted on, yet to my mind three considerations weighed with me in deciding on the Porro operation:—

DWARF.

Case of Porro-Cæsarean Section.

(1.) The operation is itself much easier and more rapid than the Cæsarean section.

(2.) It would, if successful, save the woman from any risk of a similar operation having again to be performed.

(3.) There was no object to be attained by preserving her uterus.

The simplicity of the operation and the immunity of the patient from any further trouble decided the point, and therefore a Porro-Cæsarean was performed on the 30th April last. This was, as I anticipated, simple and rapid. The incision was made from umbilicus to pubes, and the uterus rapidly exposed. The uterus was pressed up close to the abdominal wound, and made to protrude somewhat through it. I then carried a strong elastic ligature over the uterus down to the cervix. This was the only difficult part of the proceeding, and the difficulty lay in keeping the intestines out of the bite of the ligature. This ligature was then drawn tight and held so. Immediately this was secured, I made an incision in the uterus, very fortunately escaping the placenta. The fœtus was quickly extracted, firm pressure being all the while made by the elastic ligature. The child when born showed symptoms of asphyxia; but thanks to the efforts of Dr J. C. Webster, it was with difficulty resuscitated. After the birth of the child strong needles were inserted through the knot on the elastic ligatures and cervix, the uterus amputated, and the wound closed. The whole operation lasted about twenty-five minutes, and was done under careful antiseptic precautions. There was no shock whatever.

Size of Child.—Weight, 4 lbs. 6 oz.; length, 17 inches.

Diameters of Fœtal Head at Birth.—O.M., 4·75; F.M., 3·5; S.O.B., 3·5; B.P., 3·5; B.T., 3; B. front., 2·4; trach. breg., 3·5. The uterine stump dropped off on the thirtieth day after operation. Child and mother both doing well after a lapse of six months.

The last two cases to which I venture to refer are two cases of extra-uterine gestation:—

Case I.—When first seen with Dr Dickson of Newton-Stewart, presented all the appearance of a woman at full-term pregnancy; but her enormous size and the constant pains from which she suffered, as well as the history of her case, made Dr Dickson suspicious. When I saw her she was suffering from a large abdominal tumour reaching up to her ensiform cartilage, and its dimensions were larger than a full-time pregnancy. The history of her case was that six months previously she had suffered, after three months' menstrual suppression, from acute pains in her left side. Accompanying the pain there was also marked collapse, and the patient was in imminent danger for some days. From that time until I saw her she had occasionally small hæmorrhages, and now and then attacks of pain, which were most properly attributed to peritonitis.

On examination the fœtus could be felt lying obliquely across

the abdomen and enveloped in fluid. The uterine souffle and fœtal heart were plainly audible. Per vaginam the uterus could be felt enlarged and to the right side, as big as a large jargonelle pear, and separate from the abdominal mass. The diagnosis lay between a pregnancy in a bicornuous uterus and an extra-uterine pregnancy. I inclined to the latter.

She was brought into Edinburgh, and two days later labour set in in the small uterus. The pains were regular. Gradually the os opened, and the first stage of labour was complete. I introduced my fingers and found the decidua separating in shreds; gradually the labour pain increased in severity, and the decidua were shed off. Then the labour ended, being, it will be observed, an exact counterpart of the phenomena which occurs in ordinary parturition.

Three days later I operated, and removed a full-time fœtus. The details of the operation and the naked eye and microscopic anatomy I hope to publish in detail with Dr Webster later on. From the nature of the case, the death of the patient was almost inevitable.

I am indebted to the most careful and untiring work of Dr Webster for a complete knowledge of the nature of the case, which, so far as I know, is unique. Meantime, in anticipating this, I may state that the case was found to be tubo-peritoneal (partly extra- and partly intra-peritoneal).

Case II.—On the 25th of November 1890, I was asked to see M. R., who gave me the following history:—On the 26th of November 1889 she was confined of a second child easily, and made a good recovery. Menstruation returned about the third month. On the 21st of August 1890 she had a miscarriage, apparently about eight weeks. On the 22nd of September following, her menstrual period occurred apparently normal. There was no menstrual discharge in October. On November 1st there was a quasi-menstrual discharge, which was thought to be menstruation, but it disappeared in a few hours. There was frequent recurrence of this discharge throughout the month of November, coming on in the morning and disappearing in the afternoon. On the 26th of November I saw the case. There was a distinct swelling on the right side of the uterus. On the 28th an entire decidual cast of the uterus came away; slight pains were complained of. The decidua was examined very carefully, said to be complete, and considered of six weeks' formation. No ovum, however, was found. On November 29th the patient was seized about 6 P.M. with violent pains in the region of the stomach and right iliac fossa. These lasted about half an hour in a very severe form; then they abated. I saw her shortly after this, and found the swelling larger and more tender. On December 1st the severe pains in the stomach and iliac region returned. Relief was given by a large opiate. Coloured discharge continued to come at intervals. On December 12th I saw the patient after she had severe pain; there was a distinct

swelling behind the uterus, continuous with a swelling on the right side. There was pain and discomfort in passing water. The question which all along had been present to my mind was that of extra-uterine pregnancy.

From the fact of the entire escape of the decidua, which so far as my reading goes is exceptional in cases of ectopic gestation, I was disposed to think that the ovum had escaped, and that the pregnancy was intra-uterine, and that the swelling might be a hæmatocele. Still the paroxysmal pain on the right side and the slight enlargement of the swelling continued, and therefore I kept a strict watch on the patient. Gradually, little by little, the tumour on the right hand side became more cystic, more tender, and more elastic to touch. Hæmorrhage continued from time to time from the uterus, and well-marked paroxysmal pain was present every four-and-twenty hours. I did not use the vaginal stethoscope. No auscultatory sounds could be heard through the abdomen, but the enlargement of the right side tumour, gradual and definite, was a sufficiently characteristic symptom, and that superadded to the passage of the decidua and the enlargement of the uterus led me to the conclusion that the patient was suffering from an early extra-uterine gestation.

The local examination was conducted with the utmost care. The uterus, enlarged and heavy, was pushed to the left lateral and posterior side of the pelvis. The cystic mass which I have just described occupied the right side. The mass felt particularly tense and elastic, and gave the impression of being about double the size of a large orange. It was very tender and painful to the touch.

On the 26th of December, having decided that the case was one of extra-uterine pregnancy, the further interference was readily enough settled. Of course, any treatment other than that by abdominal section was not entertained. I therefore opened the abdomen on the 26th of last December, and came on a firm resistent mass in the right side. It was firmly adherent in every direction. The great difficulty was to find a place to commence the work of separation and removal. I was surprised to find the mass so fixed and so solid. From my previous examination per vaginam I expected to find it elastic and fluid, and much more mobile.

I began by ligaturing the proximal end of the tube, and after freeing the adhesions and separating the broad ligament and peritoneum, I ligatured the distal end and the broad ligament below, and so removed the mass. I left in a drainage-tube for some hours. The patient made an uninterrupted recovery. The microscopic examination must be reserved for another communication. It proved to be an extra-uterine pregnancy about the eighth week.

This case proves beyond question the possibility of deciding the presence of an extra-uterine fœtation, and removing it successfully before rupture, provided only pains and care be taken in watching the symptoms as they develop.

The President expressed the pleasure with which he had listened to Dr Croom's graphic and instructive record of his recent cases of laparotomy. It was well to have such a paper read before this Society, where they might have the benefit of the ideas of some of their surgical brethren in regard to this class of cases, which had been more frequently discussed in the Obstetrical Society. Each of the cases related by Dr Croom was of special interest, and deserving of special study. The cases of twisted ovarian tumour, for example, were well detailed, both as regards the symptoms and diagnosis and the indications for treatment. As regards the causation, he (the President) had met a couple of cases where the explanation offered by Lawson Tait quite satisfactorily accounted for the torsion; and if Dr Croom was right in maintaining that the accident occurred suddenly, he would require to show how the twists could be ten in number in the same patient, and yet the result come about so suddenly. In the matter of hysterectomy, also, he believed Dr Croom had explained the best method of dealing with the pedicle. The paper altogether was one of unusual value, and Dr Croom was to be congratulated on the brilliant successes he had achieved in this department.

Mr Caird congratulated Dr Croom on his success and his excellent paper. He thought the direction of the twist, in cases of ovarian tumour with torsion of the pedicle, was of importance in determining the factor. He was much interested in regard to the difficulties attending the small size of the bladder in cholocystotomy, since in a case of his own he had been entirely unable to find the gall bladder. The patient had constant vomiting, associated with symptoms referable to the gall bladder. An exploratory incision was made, but it was impossible, even after prolonged manipulation, to find the viscus, which seemed lost in the midst of adhesions beneath the liver. The wound was therefore closed, and the patient made an uninterrupted and complete recovery, all her symptoms having disappeared.

Dr Joseph Bell congratulated Dr Croom on his remarkable success, and had during the reading of his paper reflected on what a change in abdominal surgery the last thirty years had produced. A new world of diagnosis and treatment had been discovered. Gynæcologists had asserted their mastery over abdominal surgery, and many of the organs they had made their own.

Dr John Haddon said Dr Bell's remarks recalled to him a case in which the late Professor Bennet in 1866 asked Dr Bell to open the abdomen of a man suffering from obstruction of the bowels, in which the physician had done all he could. Then, however, it was against the principles of surgery to interfere in such a case. Now, however, the surgeon was not difficult to persuade to operate in all such cases.

Dr Bell, following Dr Haddon's remarks, remembered the case thoroughly. He had diagnosed matting of intestines from chronic

peritonitis. He had the opportunity of following the case to the post-mortem room; the diagnosis was verified, and after removing *en masse* the whole intestines, even on the dissecting-table Mr A. B. Stirling was absolutely unable to unravel the coils of intestine.

Dr Foulis could not see how the passage of fæcal matter downwards through the bowel could account for all cases of twist of the pedicle. The fæces passed always in the same direction, and might thus account for the twist in one direction. But did the ovarian pedicle always twist in the same direction? Dr Croom should examine every case of twist, so as to settle this question. As one who had followed Dr Croom's career in this department of abdominal surgery, Dr Foulis thought it right and just to say that by his great success Dr Croom had raised himself to the position of the most successful ovariotomist in Scotland.

Mr Black remarked that he had been present at some of Mr Clay's cases in Manchester forty years since, and had seen some of the more modern operations, and attributed the want of success of the older cases to their being treated in unfavourable conditions of residence in cities, and not in hospitals with their comforts and surgical aid. The introduction of the antiseptic system of operation in hospital treatment had effected a favourable change in the management of abdominal surgery and conduced to its present success.

Dr Croom begged to thank the Society for their indulgent consideration of his paper. He was specially indebted to the President for his kind remarks. With regard to the question of the twisting of the pedicle, Dr Croom thought that so far as his experience went, the cases with which he had met with twisted pedicle had been those which had emerged quickly from the pelvis and rapidly grown in size, and where being freed from the pelvis the pedicle had become elongated, and the tumour had commenced by the constant action of the intestines on the abdominal wall. He was interested in what Dr Caird had said about the gall bladder, and his case very much coincided with his own. With regard to the question raised by Mr Joseph Bell, the surgeon proper had to deal with quite a different class of cases from those who are more engaged in gynæcological work, because while the former were called in *in extremis*, the latter had the treatment of the cases from the commencement. He was very much indebted to Dr Foulis for the remarks he was good enough to make about his paper. They were to him of special value, because Dr Foulis had an exceptionally large experience in this department of medicine. He quite agreed with him that the fæces passing down the rectum by no means accounted for the twisting of the pedicle.

2. AN OUTBREAK OF TYPHOID FEVER DUE TO MILK INFECTION.

By Harvey Littlejohn, M.A., M.B., C.M., B.Sc. Public Health, F.R.C.S. Ed.

In bringing before the Medico-Chirurgical Society the account of an outbreak of typhoid fever which occurred at the latter end of September and commencement of October of last year, I do so because the epidemic, though comparatively limited, yet presented features of special interest, and also because, in a Society such as this, anything affecting the health of the city has a claim on the attention of the members and ought to be submitted to them.

A consideration of the Edinburgh notification statistics tends to show that the season of greatest prevalence of typhoid fever is during the three autumnal months—September, October, and November. This coincides generally with the statistics of other places in the United Kingdom, and also with Buchan and Mitchell's curve, based on the mortality returns of London. In fact, typhoid fever remains low during the first six months of the year, commencing to increase in August, and reaching the maximum in October, after which it rapidly diminishes.

During 1890 the number of cases of typhoid notified in Edinburgh each month was as follows:—

Jan.	Feb.	Mar.	Apr.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
17	22	27	21	24	16	29	20	24	79	43	178

These figures are shown on the Chart, and are contrasted with the monthly averages of the previous ten years.

The Chart, although showing no great reduction in the number of cases of typhoid during the first six months of last year, yet gives no evidence of any tendency to rise in August and September, as one would naturally have expected. Indeed, the number of cases notified during these two months is actually lower than in the corresponding months of the last five years, with the solitary exception of 1888. From this, then, it is seen that up till the commencement of October there was not an unusual number of cases of the disease in town, and, in fact, during the previous two months these had been unusually low. During September, however, as I shall show, the germs of typhoid were being insidiously spread, with the result that during the first eight days of October no fewer than 25 cases were notified to the authorities, and by the end of the month there was a total of 79, causing the abrupt rise seen on the Chart. It is to this outbreak I would direct the attention of the Society to-night.

It has been stated that during September there had been comparatively few cases of the disease notified, but among them were two deserving of special attention, and which occurred in *one* family towards the end of the month, viz., one on the 25th, and the other on the 27th, on which latter date both were removed to the Fever Hospital. The doctor in attendance on these two cases had at first some difficulty in coming to a diagnosis, and even after the nature of the illness was apparent, he was unaware that both patients were employed in a milk-shop. The result of this was, that not only did the doctor remain ignorant of the fact, but also the authorities, until some days after their removal into Hospital. On September 30th, however, on the occurrence of two other suspicious cases in the neighbourhood, a visit was paid to the milk-shop. It was then discovered that the two patients above mentioned had been actively engaged in the work of the shop when they were seized with the fever. No cause could be adduced to account for their illness, either at their home or at the shop; but it was incidentally mentioned that both were very fond of milk, and drank large quantities; while a third sister, also employed in the shop and quite well, scarcely took any milk at all.

On finding no sufficient cause for the origin of the cases in the sanitary state of the milk-shop, the milk supply was inquired into, and the information elicited that milk was obtained from three separate dairy farmers, viz., one situated quite close to the town, and the other two at some distance in the country. These farms were thereupon visited, and two of them were found to be quite satisfactory. At the third, however, I found in the family of the dairyman one of the children in bed, and evidently ill. On inquiry, the mother said that the child had only bronchitis, and was almost convalescent, and therefore she had not thought it necessary to call in the doctor. On examination the tongue was found to be coated, the temperature slightly raised, and the general appearance of the child not quite consistent with an ordinary attack of bronchitis. The mother stated that there had been no diarrhœa except what had been produced by a dose of castor-oil. The house and inmates were very dirty, and the appearance of the farmyard highly unsatisfactory. The mother was told that the doctor must be called in at once, and at the same time I wrote to him, stating my suspicions, and requesting him to telegraph his diagnosis of the case as soon as possible. On the following afternoon the information came that, in his opinion, it was typhoid, and that the patient had at once been removed to another house at some distance from the dairy. Immediately on receipt of the telegram steps were taken to have the sale of milk from the farm stopped in the town, and for this purpose an order was applied for under clause 211 of the "Edinburgh Municipal and Police Act, 1879," which states that —"When it shall be certified by the medical officer of health to the chief constable that milk is being brought within the burgh from

any farmhouse, dairy, or other place beyond the burgh in which any person is ill of any infectious or contagious disease beforementioned,[1] and that the said milk is being sold or offered for sale in any shop or premises within the burgh, or distributed therein from carts or otherwise, the prosecutor shall apply to the judge of police for an order to prohibit the sale of milk so brought from and after the intimation of such an order until the person affected has been removed or shall have recovered from such illness, the premises been disinfected, and the bedding and clothing have been destroyed or thoroughly disinfected, and it is certified by the medical officer of health that the said premises are free from infection, and that the sale of milk from such place may be safely resumed." Unfortunately the machinery of this clause works slowly, and it was found impossible to get a *legal* prohibition put on the sale of the milk till Monday the 6th of October. On Friday the 3rd, however, all the dairies and customers known to be supplied with the milk had been warned of the danger, and an assurance was received from them that they would discontinue taking in the supply for the meantime. In cases where some of the suspected milk was found still unsold in shops, this was bought up by the authorities and destroyed.

It can, therefore, be assumed that the supply of milk from this farm was practically stopped on October 4th, and that none of it was sold in the city after this date until the subsequent withdrawal of the police order.

It may appear as if the decision to regard the milk with grave suspicion and to prevent its sale was premature, considering the great injury it involved to the milkseller should our conclusions prove to be ill-founded, and his business in consequence be practically stopped. No doubt it was the time of year when a considerable increase in the number of cases of typhoid fever was to be expected, and this, too, occurring in a manner which might give rise to a suspicion of some common source of infection. The coincidence, however, of the cases of fever at the farm and dairy-shop, and particularly the vivid impression left on my mind at the first visit of the extremely insanitary surroundings of the farm, gave one an intuitive feeling that in some way the farm was closely connected with the two cases which occurred at the milk-shop in town. Justification of the action of the authorities was, however, not long in being forthcoming.

On October 2nd five intimations of typhoid fever were received, on the 3rd five, on the 4th four, until during the first eight days of October no fewer than 25 cases had occurred, to all of which milk from this farm had been supplied.

I have given a short account of the manner in which attention was first of all attracted to the outbreak, and how the milk came to be regarded as the probable source of infection.

[1] Small-pox, cholera, typhus, typhoid, scarlet fever, diphtheria, measles.

After that, one naturally began to look back among the cases of typhoid reported during the previous few weeks, before suspicion was excited, for any which might be ascribed to the same cause.

During September 24 cases of typhoid were notified, and on inquiry it was found that in 8 cases occurring in different families milk was obtained from milk-shops which received at least a portion of their daily supply from the suspected farm. It will, probably, be here remarked by some that if, from the first, inquiry had at once been made into each case as it occurred, and its milk supply ascertained, the subsequent outbreak might have been *wholly* averted. This is partly true, but I would point out that these 8 cases occurring in September were for the most part widely distributed over the town, received milk from different dairies, and had no apparent connexion with each other. Thus, 1 case was situated in Buccleuch Street, 2 in Lauriston Place, 4 in Morrison Street, and 1 at the end of Dundee Street.

It is the practice of milksellers in Edinburgh to get their milk from several different wholesale dealers, I suppose for the purpose that no accidental interference with one of the supplies may affect their retail business. Hence, in order to trace the connexion between the above cases, it was not sufficient merely to get the name of the milk-shop, but also to find out from what farms the supply was drawn. It was then found that the milk from the suspected farm was distributed to the following milk-shops in town (see Map):—

I. and II. in Morrison Street.
III. in Fowler Terrace.
IV. in Tollcross.
V. in West Bow.
VI. in Buccleuch Street.
VII. in Salisbury Place.

But in addition the dairy farmer delivered milk directly from his cart to three houses (VIII.) in Warrender Park Road, to four families in one tenement in Riego Street, and to a family in Bristo Street.

I have already mentioned that of the 24 cases notified in September, 8 received milk from one of the above milk-shops. The dates of their occurrence were as follows:—

Sept. 23, 2 cases.	Sept. 26, 1 case.
„ 24, 1 case.	„ 28, 2 cases.
„ 25, 1 „	„ 30, 1 case.

Let us continue to follow the progress of the outbreak:—

On Oct. 1st, 6 cases were notified, 5 with the infected milk supply; and of these, 3 occurred in Gardner's Crescent, 1 in Fowler Terrace, and 1 in Grange Road.

On Oct. 2nd, 8 cases were notified, 5 with the suspected milk

supply, and distributed as follows,—1 Buccleuch Terrace, 2 Rosemount Buildings, 1 Gardner's Crescent, and 1 Hazelbank Terrace.

On Oct. 3rd, 4 cases were notified, all getting the milk—3 of them in the Bryson Road and 1 in Earl Grey Street.

On Oct. 4th, 4 cases, 3 getting the milk—1 in Glen Street, 1 in Rosemount Buildings, and 1 in Gardner's Crescent.

On Oct. 5th, 2 cases—1 in Rosemount Buildings and 1 in High Riggs—both getting the milk.

On Oct. 6th, 3 cases, 2 getting the milk, and both in Rosemount Buildings.

On the 7th, 2 cases, 1 getting the milk, in Gardner's Crescent.

On the 8th, 4 cases, 3 getting the milk—2 in Gardner's Crescent and 1 in Johnston Terrace.

And now there was a lull in the number of cases reported, at least so far as those getting the suspected milk were concerned.

On the 14th, however, 8 cases were notified, and 5 of these were found to be getting the milk, viz., 2 in Marchmont Road, 1 in West Bow, 1 in Cobden Terrace, and 1 in Hope Park Crescent.

On the 15th and 16th, 1 case on each date was notified, and after this cases appeared pretty regularly up till the end of the month, for the most part in the streets and buildings already mentioned.

During November 43 cases of typhoid were notified, and of these 6 were connected with the above milk or with previous cases in the same house.

In all, there were 63 persons who were undoubtedly affected with the disease, and afterwards, in giving some statistics of the outbreak, I shall only refer to these 63 cases. But in addition, during the same period there was a large number of cases of undefined illness—of malaise, of severe "bilious" attacks, seizures of vomiting and purging—some attended by medical men, some not, but all receiving the same milk supply, which, in the light of our present knowledge, may almost certainly be identified with the above cases of typhoid and their origin traced to the same cause. Accidentally when conducting other inquiries, and even when investigating lately other cases of typhoid, we have been informed of cases of illness which occurred about the end of September, the symptoms of which quite tally with those of mild cases of typhoid, and in some families subsequent undoubted and notified cases may be traced back in their origin to these primary illnesses.

To return, however, to the 63 cases above referred to, some interesting details may be mentioned regarding them. There were 25 males and 38 females affected. The ages of the patients afford striking proof that in typhoid it is not the children who are at home, and who are supposed to drink a large quantity of milk, who are most affected, but adults, young men and women, who are perhaps out all day, and only take breakfast and supper at home.

How can one account for this somewhat anomalous fact? I think that whatever other factors there may be, the conditions which chiefly influence the incidence of the disease as due to milk infection so far as age is concerned appear to be—(1), the age of greatest prevalence of the disease, viz., in typhoid about 20; (2), the quantity of milk consumed; (3), the health of the person at the time of infection, more especially as regards bodily vigour or fatigue. Children, with few exceptions, are rarely without food for any length of time, and thus the stomach is seldom empty. Neither are they often in a state of great bodily fatigue. Young adults, on the other hand, go out to their work early, come home tired and hungry, and if they are milk drinkers, they take a tumblerful or more of milk straight off. In a large number of the cases I have satisfied myself, both from inquiries and voluntary statements, that the disease occurred in persons who, to employ an expression used by many, "lived on milk," or were very fond of it and drank considerable quantities every day. On the other hand, in several of the cases I could only discover that patients had taken milk along with tea or coffee, and therefore in very small quantity.

In classifying the cases according to age, the rough divisions on the Table have been taken, where the first division may be held to

1–5 Years.	5–15 Years.	15–30 Years.	30 — Years.
7	16	31	9

include infants, the second school children, the third young adults, and the fourth middle-aged persons. From this it will be seen that almost 50 per cent. of the cases occurred in young adults.

In regard to the members of households affected, it was found that in fourteen families containing children *only adults* were affected, and some of these afforded very striking examples of the selective power of the poison. For instance, in a family of three adults and six young children, who all partook regularly of the milk, the youngest adult, a male, 25 years of age, alone was affected. This patient used to be out all day working hard, and was in the habit of taking one or two tumblers of milk every evening for supper. In eight families *only children*, or persons under 15 years of age were affected, and in twelve families both adults and children were struck down. In all, forty-one households were affected, giving the 63 cases above mentioned.

1 case occurred in 26 houses.
2 cases „ „ 11 „
3 „ „ „ 1 „
4 „ „ „ 3 „

The mortality was low, which corresponds with the mild type of the disease. There were three deaths, equal to a mortality of 4·7 per cent. The ages of the three fatal cases were 22, 29, and 17, and all were females.

Unfortunately, I am unable to offer reliable statistics as to the numbers infected in relation to the total number of persons exposed to infection. This is chiefly due to the fact that at most of the milk-shops it was impossible to obtain a complete list of customers, a considerable quantity of milk being sold to persons who were either unknown to the retailer or not regular customers of that particular milk-shop. Thus, not only were some of the customers not known by name, but they, to a certain extent, changed from day to day. Another circumstance which prevents an exact estimation of the number which received the milk consists in the fact that on some days the milk from the suspected farm was sold by itself unmixed, while on other days, depending purely on chance, it would be mixed with an equal or larger quantity of milk from other sources, and thus have a much wider distribution, though in a more dilute form as regards infective material. For these reasons, the tracing of the milk to certain regular customers was rendered practically impossible, and has prevented me from giving a comparative table of the numbers supplied by each milk-shop and the numbers affected with the fever.

The circumstance, however, of this particular milk having been sometimes sold by itself, at other times mixed with varying quantities of milk from other dairies, goes far to explain the extraordinary limitation of the disease among the customers of certain of the milk-shops. It does not, however, by any means fully do so. For how are we to account for the fact that milk-shop I., receiving 4 gallons of sweet milk and 2 of skim per day, is accountable for no less than 25 cases; while II., getting 6 gallons sweet and 6 gallons skim, is accountable for only 6? Again, III. got 6 gallons sweet and 4 of skim, and supplied 11 cases, IV. 3 gallons and 8 cases, V. 12 gallons and 2 cases, VI. 12 gallons and 3 cases, and VII. 3 gallons and 1 case. These figures are somewhat bewildering in their apparent inconsistency.

At present we know too little about the laws governing the incidence and conditions of attack of typhoid fever to be able to give the key; but in all probability there were certain days when the milk was contaminated more than others, and this is easily accounted for if the typhoid poison was contained in the water at the farm; or again, the fact of the milk being largely diluted with other milk on some occasions, on others being sold unmixed, together with the varying daily quantities sold to each family, are all factors which must undoubtedly be taken into account in the solution of the difficulty. From a rough calculation, not by any means accurate, I think it quite possible for the milk, in a more or less pure state, to have been distributed to fully

400 families, or, giving 5 individuals to each family, to 2000 persons.

What was the cause of the epidemic? Were the cases due to infected milk, and if so, how was the milk contaminated?

In the first place, I think it will be readily admitted that, notwithstanding the fact that October is the month when one expects an increase in the number of cases of typhoid, in the present instance we were dealing with something distinct from the regular autumnal increase, and which must therefore have been the result of unusual conditions. As has already been shown, there had been comparatively few cases of the disease during September (namely 24), when suddenly during the first week of October there were no less than 25 intimated to us. This is no steady and gradual increase, such as one under ordinary conditions expects; it is an outbreak, and must therefore depend on a special cause. The causes which might be common to a number of cases spread over a considerable area are, the milk and water supplies and the state of the drainage, and therefore, before we can prove the theory of infected milk supply, we must be able to exclude the other two, viz., water and drains. The water is easily disposed of, because from the wide distribution of the cases it was at once found that none of the city supplies was common to the various cases, and that water from different sources was being distributed to the infected houses.

In regard to the drains, however, it is more difficult to disprove their connexion with the cases, and this is only as one would have expected, since it is impossible to examine forty or fifty houses in any town and not find in many of them sanitary defects, which with the presence of a case of typhoid in the house would not be deemed amply sufficient to account for the disease, but which, on the other hand, *without* any history of illness, would be merely regarded as small imperfections of no great moment.

In the present instance, the exclusion of the drains as the cause of many of the cases is rendered even more difficult from the undoubted insanitary conditions which obtained in many of the houses. I do not refer so much to grave defects in structure, as to the want of water which existed during the summer in a number of the houses in which cases occurred. More particularly this want of water existed in Rosemount Buildings, a densely populated block of workmen's houses, supplied by milk-shops I. and II., and furnishing a considerable number of the total cases of the disease. The water was never off for more than six hours during the day, and even then a supply could always be obtained from a tap in the centre of the square. Notwithstanding this, however, there was at the time a decided scarcity, with the result that W.C.'s were insufficiently flushed, and smells were complained of. All the houses where cases of fever occurred have been examined by the Burgh Engineer, and with few exceptions the sanitary

fittings were found to be in good order. It may be further stated that in personally visiting every case I made a point of asking the people if they had any complaints to make as to smells or water supply, and almost invariably, except in some of the houses in Rosemount Buildings, received a negative reply.

In proof, too, of the fact that the drains, although not wholly satisfactory in some instances, yet had not been a source of disease in the past, may be adduced the fact that in only three of the houses at present under consideration have cases of typhoid occurred during the last six years. This is a striking fact. These houses were not fever dens, were not already polluted dwellings, in the drains of which germs of the disease had been lurking from previous cases: they were houses remarkable for their freedom from the disease.

The strongest arguments against the fever having been due to drains, however, still remain to be mentioned, and these are, the manner in which the cases occurred, their distribution, and lastly, the extraordinary fact of their all receiving milk from milk-shops supplied by a certain farm in the country.

If bad sanitary arrangements had been the cause, we should have expected the cases to have cropped up gradually, and to have been localized to one drainage area,—not for them to break out with sudden intensity, and to be spread over a very extensive district, stretching from Buccleuch Street in the east to North Merchiston in the west.

And, lastly, against the view of infection from drains is the evidence directly in favour of the milk theory.

With a comparatively small number of cases during September (24), suddenly during the first eight days of October no less than 25 cases are reported, and during the rest of the month intimations continue to come in, till at the end of October a total of 79 cases have been reported, a number large even for October in a bad typhoid year. But when on inquiry it is found that a large majority of these cases, distributed over a wide area, and having apparently nothing else in common, get their milk from seven separate and distinct milk-shops, which, however, all receive a portion, at least, of their milk supply from a certain farm in the country, and when on a visit to this farm a member of the family is found to be ill and suffering from typhoid fever, I think there can be no doubt in the mind of any reasonable man that in some way or other the milk had been intimately connected with the outbreak of the disease. I would be inclined to go even further, and say that even if we had not found a case of illness at the farm, even if the sanitary arrangements there had been found satisfactory, yet the extraordinary coincidence of these cases spread over such a wide area occurring at the same time, and all receiving milk from one source, is of itself practically conclusive of a milk infection.

If any further proof of the part played by the milk in the

present instance were wanted, it could not have been better furnished experimentally than by the following circumstance. I have already stated that the milk from the farm was supplied to seven milk-shops, and that the man supplied directly from his cart seven families in three separate houses in Warrender Park Road, four families in one house in Riego Street, and one family in a house in Bristo Street.

There had been no cases of typhoid for a long time in the houses in Warrender Park Road and Riego Street, and it was therefore with considerable interest we awaited the progress of events. If the milk was contaminated, and that at the farm, then we should expect in at least one of these houses a case of typhoid to occur. Our fears, or perhaps one should say hopes, were fully confirmed. On October 14th two cases were intimated from one of the houses in Warrender Park Road, and on the 28th a case in another of the families supplied with the milk from the cart, and subsequently cases were found to have occurred in the house in Bristo Street, in each of the three houses in Warrender Park Road, and in one of the four families supplied in Riego Street.

After this striking evidence I think we must admit the rôle played by the milk, and now let me say a few words on *how* the milk became infected. In attempting to explain this, it is necessary to examine closely the sanitary conditions which existed at the farm on the date of my first visit. The farm is situated on sloping ground, consisting of loose gravelly soil. The farm buildings are of considerable size, and part of them are let to a dairy tenant, who has his own servants, and has otherwise no connexion with the farm proper. In Fig. 2 a rough diagrammatic ground plan of the buildings and surroundings is given. The block E, B, F, G is that which was occupied by the dairy tenant, and with which, therefore, we have specially to do. B represents the byre, which contained twenty cows. It is a dark, low-roofed building, the floor being paved with small rough stones, with wide interstices between them. The whole byre was extremely dirty, and the surface covered with manure, solid and liquid, due in great measure to the extremely deficient drainage arrangements, or perhaps it would be nearer the truth to say the absence of any efficient drainage. F is a small room opening directly off the byre, and at a lower level than it, which served apparently the double purpose of a scalding-house for the cans and a place for keeping and filling them after the milking, besides acting generally as a place for ordinary washing operations. The floor consisted here also of small rough stones, and in one corner was a grating which was supposed to carry off the waste water from a pump, and was directly connected with the rough drain for carrying away the liquid manure from the byre to the dungheap K. This drain was composed of badly-jointed, rough pipes, which, it is needless to say, were as often choked as not. G is the milk storeroom, and except for its

surroundings was fairly clean. C is a somewhat roughly constructed pig-stye, containing several pigs, and kept in a very dirty condition. K is a manure heap, containing a large quantity of both solid and liquid manure. The road in front of it, and leading up past the byre, was very dirty and badly drained. A is the farmyard, and here also there were several pigs and a large quantity of manure.

In E, the dwelling-house, there lived Mr and Mrs B., their six children, and two servant girls. Mr and Mrs B., these two girls, and a boy who did not live on the premises, were all who had anything to do with the dairy work. Mr B. and the boy drove the milk into town, while Mrs B. and the two girls, besides their duties in connexion with the dairy, had also to attend to the household work and children. The house was dirty and untidy, which was only to be expected considering the numerous young family and the time and labour required in connexion with the management of the dairy.

The sanitary arrangements of the house were of a somewhat rude description. There was no privy, ashpit, or place for getting rid of refuse in connexion with the house. All excrement, etc., had to be carried past the byre and milk-house to the dungheap K, so that it can easily be imagined, where the disposal of refuse was attended with such inconvenience, that the children and others would be encouraged rather than otherwise either to dispose of it nearer the house, or to attend to Nature's call in the first and most convenient spot that could be found. The water supply was no less primitive. There was no supply led into the house; it had all to be carried from a pump in the scalding-house F. This pump was connected by a leaden pipe with the well D, situated in the adjoining field. This well was the sole source of water supply to the house and for all dairy purposes, including the watering of the cows. It was 14 feet deep, with sides constructed of rough, uncemented stones, between which water could be seen percolating at various depths from the surrounding soil. The top of the well was covered by a stone, and was on a level with the surface of the surrounding field, which had been transformed into a sort of quagmire by the cattle coming to drink at the adjacent trough.

The general slope of the land was from the byre to the well, and (see Fig. 1) it seems almost certain that the surface water from the farmyard, byres, piggeries, and even dungheap K must have found its way to the well. This would take place with all the greater facility when, as Mr B. stated, the well sometimes became very low, and when, as is well known, its radius of drainage influence would be much extended. Mr B. stated frankly that frequently the water had a smell, and was coloured so as to raise doubts in his mind of its wholesomeness. It would certainly appear as if all possible means of contamination of the well had been already mentioned, but Mr B. informed me that he was in

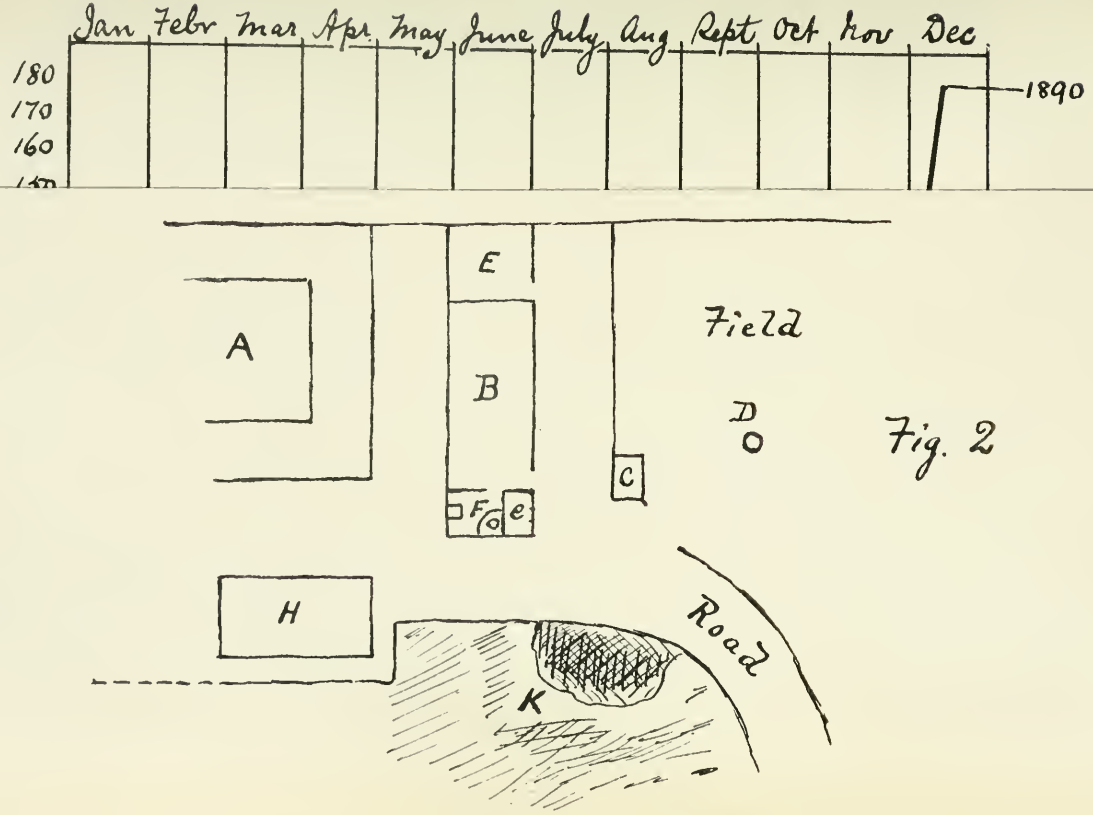

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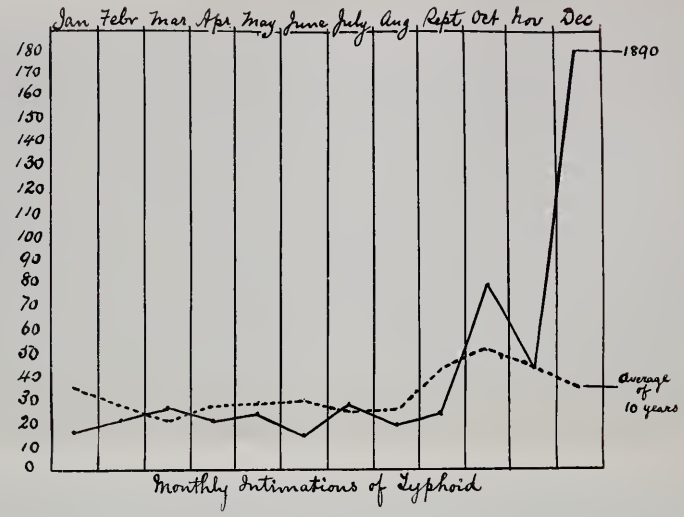
Fig. 2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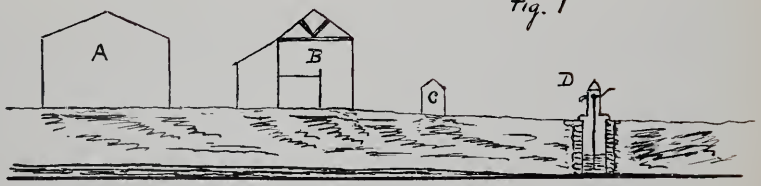
1890
Average of 10 years
Monthly Intimations of Typhoid

Fig. 1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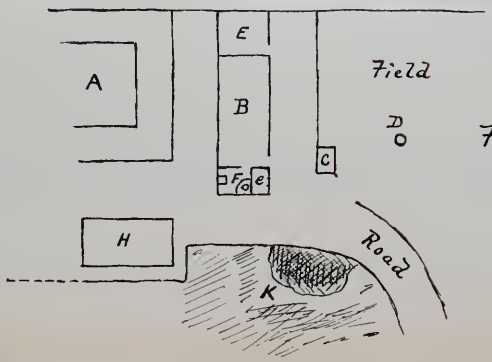
E
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Fig. 2

the habit of pumping up liquid manure from the dungheap K and pouring it on the field surrounding the well, so that here we have actually another and, if possible, more dangerous source of contamination of the well water.

An analysis of the water by Mr Falconer King, the city analyst, showed the presence of a large amount of alkaline nitrates and chlorine sufficient to render it dangerous and unfit for any dietetic purpose. No biological examination was made.

Such were the conditions which existed at the farm, and it must be acknowledged that in most respects they reveal a state of matters as highly undesirable in connexion with a milk supply as could well be imagined. The badly drained and filthy condition of the byre and scalding-house, the insanitary surroundings in which the milk was daily manipulated,—a circumstance too often lost sight of,—the polluted water used for washing the cans and for all domestic purposes, and, lastly, the want of cleanliness in the family of the dairyman, are all factors to be taken into account in considering the question of how the milk became contaminated. According, however, to the present state of our knowledge in regard to the origin of typhoid fever, these conditions are not sufficient in themselves to account for the outbreak. This consideration led us to make minute and careful investigation into the health of all connected with the dairy-farm during the previous summer. Mr B., it was found, was ill for a week or two during the beginning of August, but was never confined to bed, and according to his own statements and that of his medical attendant, he exhibited no symptoms at all resembling typhoid fever. *No history of illness could be obtained as having occurred in any member of the family or other person connected with the dairy during the summer*, and this statement was corroborated by the doctor who was the usual medical attendant. Inquiry was made as to any friends who might have visited them during the summer months, but likewise with a negative result.

The first case of illness which occurred at the place was that of Mr B.'s child, who was discovered in bed on the occasion of my visit on October 1st, and she first complained of illness during the last week of September, that is to say, several days after cases of typhoid had been reported to us in town, among persons receiving milk from this farm. The first of the cases in town which I attribute to infection from this farm took ill on September 14th, which would date the infection to about September 1st in all probability, and hence I think we must regard Mr B.'s child as one of the victims, and not as the primary cause of the outbreak. Notwithstanding every effort, no case of illness to account for the infection of the water or milk with typhoid germs previous to this case could be discovered, and hence I fear this outbreak can only add another to the already long list of epidemics which have been traced to milk infection, but in which the last link in the chain of evidence, viz.,

a first or original case, could not be discovered. Subsequent to the case of the little girl above mentioned, another of Mr B.'s family was seized with the disease, and also a boy who worked at the farm, but not in connexion with the dairy, and who lived at some distance off, was taken ill and died of the disease. This latter patient used to drink the water from the well, but otherwise had nothing in common with the members of Mr B.'s family.

No other cases occurred either at the farm or in persons who were in any way connected with it.

Had time permitted, I would have liked to discuss some points connected with the periods of incubation in the various cases and the probable date of the first contamination of the milk, and also certain questions in regard to the relations between towns and their milk supply which this outbreak brought into prominence, but these I must leave for a future paper.

In conclusion, let me repeat briefly the evidence in favour of milk being regarded as the cause of the epidemic.

1. Cases arise simultaneously among customers of all the milk-shops in town and in certain private houses supplied directly from the farm.

2. A case of typhoid is found at the farm, and two others subsequently occur there.

3. The sanitary condition of the farm is exceedingly bad, and the filling of the milk cans is carried on daily in a foully tainted atmosphere.

4. The water supply is found to be contaminated by sewage and liable to gross pollution.

5. The milk supply was stopped until a better water supply had been provided, after which no more cases occurred.

Description of Charts and Diagrams.

I.—Chart showing monthly intimations of Typhoid during 1890; also average monthly intimations of Typhoid during ten years, 1880–1889.

Fig. 1.—A, Farm steading; B, Byre; C, Piggery; D, Well.

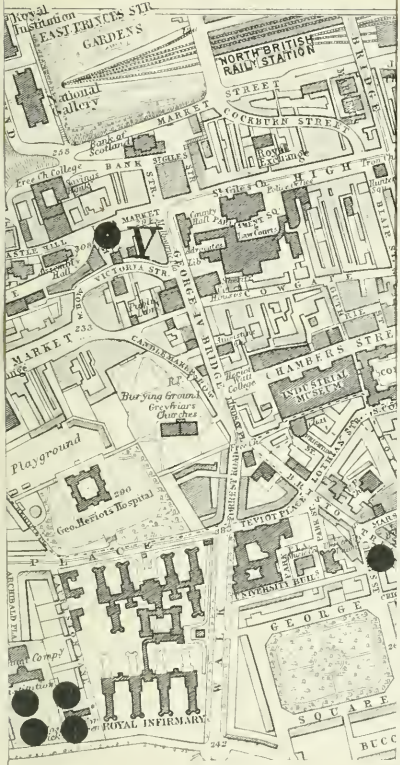
Fig. 2.—A, Farm steading; B, Byre; C, Piggery; D, Well; E, Dwelling-house; F, Scalding-house and pump; H, Cottage; K, Dung-heap; e, Milk-house.

II.—Map showing distribution of cases in the town thus ●, with milk-shops marked by Roman numerals.

The President believed he expressed the impression made on the minds of all the members by Dr Harvey Littlejohn's interesting paper when he said that the author had convinced them that he had traced out very clearly the source of the recent epidemic of typhoid. He (the President) thought there was no room left for doubt that the farm whose insanitary conditions had been so distinctly demonstrated was the breeding-place of the mischief.

Surgeon-Major Black observed the insanitary condition of the

BUTION OF TYPHOID CA



Map illustrating Dr. Harvey Littlejohn's Paper "Distribution of Typhoid Cases"

farm supplying the milk to the houses and families in the city was a fair type of country farmsteads conducted and maintained under old-fashioned notions and uninstructed in modern sanitary science. He had visited the sewage works and farm of a Midland city some years since, and looked over the stables and byres on it, which he found in an impure condition and insanitary management, though worked under the authority of the Corporation. The knowledge of sanitary science, therefore, requires yet to be extended much more amongst agriculturists and citizens in towns to secure pure food and healthy lives.

Dr Haddon said he congratulated Dr H. Littlejohn on the ingenuity and perseverance he had displayed in tracing the sudden increase of cases of typhoid fever, which took place in October, to the milk supplied by the suspected farm. He had brought together a large amount of evidence, which seemed to fix upon the milk from the farm in question as in some way connected with a considerable number of the cases that occurred; nevertheless, there were some points on which it would have been well if further inquiry had been made. Dr Littlejohn in his paper had rightly said that in an inquiry of this kind we should first of all be sure that neither the drainage nor the water-supply could be held in any way responsible; but he exonerated the water-supply, because he found the cases occurring in districts supplied by water from different sources. That consideration might be sufficient to exonerate the water as a possible source of the disease, were we sure that there was no possibility of it being polluted about the houses in question. On this point, however, there was room for doubt, since in his paper he mentioned that in one of the localities, at least, they had been short of water, and the supply had been intermittent. Intermittence in a water-supply was in itself a source of danger, and in the present case might have to do with the fever met with. Again, as to the sanitary state of the houses themselves, all Dr Littlejohn could say for them was that the Burgh Engineer reported them to be in a *fairly satisfactory* condition. Now, it was well known that the sanitary state of Edinburgh generally was far from what it should be, and if the houses in which the typhoid cases occurred were only in a fairly satisfactory sanitary state for Edinburgh, there was a probability that defective sanitary arrangements might have to do with the disease which was found in them. Again, it appeared that the dairies which were distributing this milk were not blamed for producing typhoid cases in direct ratio to the amount of milk they received from the suspected farm, but rather in an inverse ratio, since those receiving most milk were blamed for few, while those receiving least were credited with causing most cases. That fact was difficult of explanation. Again, it was the adults who were mostly affected, those, viz., who were absent from home, being engaged elsewhere during the day. It would have been well if the nature of their employment had been ascertained, as well as

whether they drank any water or milk when away from home, which might have been polluted. Again, forty-three cases are said to have occurred in November, only six of which could, either directly or indirectly, be traced to the sufferers from the suspected milk. How did the others arise, and how can the far greater number that occurred in December be accounted for (when the milk-supply had been cut off), unless either the sanitary arrangements were defective or the water-supply contaminated? Further, we must never forget that neither typhoid fever nor diphtheria are ever absent from Edinburgh, so that we need not be surprised if we meet with occasional outbursts of these diseases, without the introduction of infection from beyond her boundaries. Such considerations as these ought to make us careful in submitting our conclusions to the public, unless they are supported by the most convincing proofs, lest we should be put down as faddists, and so retard instead of advancing the progress of sanitary science among the people.

Dr Webster thought that in regard to the origin of this epidemic of typhoid fever, as practical men they must not overlook the well-authenticated fact, that the foul state of drains generally in premises, with the presence of the typhoid poison, did not necessarily cause an outbreak of the disease among the occupants. There was a certain immunity slowly produced. They might take it, but a stranger visiting was at once struck down with the disease. The analysis of the well water showed contamination, not only or chiefly of recent date. Such a foul state of drainage could not be laid to the Local Authority of Edinburgh. The city had nothing to do with it. The new Local Government (Scotland) Act, 1889, in the provision of county medical officers, would prove a boon. The county medical officer would correct such outrages on the laws of health, and would find more than enough to occupy his whole time without being crippled with private practice. This unfortunate occasion was met promptly and well by their medical officer of health. The numerous cases, since the outbreak, during the winter, long after the stoppage of the milk, were no doubt connected with it, and due to defective housepipes, traps, and other sanitary irregularities in their houses. They were the "distant" cases of the epidemic, so acutely investigated and graphically described by the reader of this important paper.

Dr Foulis had long since come to the conclusion that milk was by far the most common vehicle by which the poison of typhoid fever, of scarlet fever, of diphtheria, and such like diseases, was spread among us from a given centre of infection. Dr Littlejohn, in his paper to-night, had in the clearest way shown that the milk was the vehicle by which the poison of typhoid fever was distributed among many persons residing in different parts of the town at one and the same time. In proof of this we had simply to follow the milk-cart from the infected farm on its rounds as it left the milk

at various houses. While it was thus perfectly certain that the milk conveyed the infection, it was not so clear how the milk itself had become infected. Though the well-water was in a horribly putrid condition, Dr Foulis did not think it had been clearly shown in the paper that this water was without doubt the cause of the milk infection. In itself, the water, as shown by the chemical analysis, was putrid and quite unfit for drinking purposes, and one might say that it was bad enough to cause typhoid fever in those who drank it; but we could not overlook the fact that this same water had been in use for a long time before the present outbreak of typhoid fever. Had the girl, who was said to be suffering from typhoid fever at the farm, anything to do with the contamination of the well-water? Did her typhoid secretions in any way affect the well-water? Or was she one of the victims of the milk infection? There is not the slightest doubt that the milk conveyed the infection from the farm to numerous persons in Edinburgh, but Dr Foulis did not think it had been demonstrated how the milk itself had been infected, though the evidence was very strong that the well-water was the cause. Within the last few years a great change had taken place in our knowledge of these epidemics. A few years ago, so mysterious were these epidemics that our theological friends used to teach us that they were sent to us by Providence to punish us for our sins. Now, however, we refused to listen to such nonsense, and were doing our best, one and all, to prevent and check such epidemics; and, like the poet of old, we cry, Happy are we who know the causes of things. The truth is, most of such epidemics are entirely preventible, but, unfortunately, we have to contend against ignorance, carelessness, and wilful deception on the part of those who supply us with milk. Hence the difficulty in fighting against infection under the present arrangements. All medical men within the city boundaries are obliged to intimate to Dr Littlejohn the existence of infectious cases under our care; and here let me beg of my medical brothers not to delay a moment longer than is necessary in sending in such intimations, for it is most important that the Officer of Health should have all such cases brought under his notice without delay, in order that he may see if there is any direct cause for such cases; and let us remember, too, that the power to stop the milk-supply at one infected centre rests with one man, viz., our Officer of Health. Hence the importance of giving him all the information in our power. For it is only by such information that he is enabled to act quickly and decidedly in stopping the supply of infected milk. Regular and frequent inspection of farms and dairies inside and outside the city boundaries must come sooner or later. In this way alone shall we be able to check such epidemics at the beginning. It is rather too late to stop the milk-supply after sixty or more cases of infection have turned up. Unfortunately every important change in our sanitary laws comes very slowly, and what can we

do in the meantime to protect ourselves and our children from the evil consequences of drinking poisonous milk? We have only to cook or boil such milk thoroughly before we swallow it to make it perfectly healthy. For three years past, in Dr Foulis's house every night at 7 o'clock the cook boils for at least ten minutes half a gallon of milk in a water bath. The latest scientific experiments in Germany have shown that the poison or the organism of typhoid fever is entirely destroyed by keeping such infected milk at a temperature of 80° C. for five minutes. A temperature of 212° F. makes the destruction of the poison doubly sure. No one should drink uncooked milk unless the source of such milk is known to be pure. Milk boiled in a water bath for ten minutes has no disagreeable taste or smell, and what a comfort to know that it is perfectly free from all poisonous properties. Dr Foulis believed that an enormous amount of mortality among children in all stations of life could be prevented by thus cooking their milk food.

Dr Smart recalled the attention of the Society to the specific objects contemplated by Dr H. Littlejohn's inquiry into the cause of this particular outbreak of typhoid fever. It was not, as a preceding speaker supposed, an endeavour to explain the existence of typhoid fever in Edinburgh at the present, or at any other time; nor was it to show that at a certain dairy-farm the insanitary surroundings, and especially the water-supply, were of a nature to give rise to typhoid fever, or that these insanitary conditions did actually originate the case of typhoid in the girl which Dr H. Littlejohn found at the dairy-farm. The purpose of the investigation, as Dr Smart understood it, was to connect a specific outbreak of typhoid fever with a particular milk-supply distributed from a certain dairy-farm, the milk probably becoming infected by the discharges of the typhoid patient staying at the farm, and in some way reaching the water-supply, and through it contaminating the milk either by the milk-cans being washed in the tainted water or the tainted water being added to the milk. The question, how this girl with typhoid, staying at the farm, contracted the disease, is not raised, the patient probably being the victim of typhoid existing elsewhere. Dr H. Littlejohn, in carrying out his most interesting and important investigation, had, at every stage of the inquiry, convincingly demonstrated the coincidence in every case of an outbreak of typhoid fever in the houses to which the milk was distributed over districts quite apart, and over a widely extended area. These fever cases, occurring at the normal time of their incubation after receiving the milk, and showing the normal rate of progression of typhoid fever, afford corroborative proof of their being connected with that particular milk-supply; as also, the stoppage of the suspected milk being followed by the cessation of the further growth of the epidemic. Dr H. Littlejohn, in practically carrying out this great investigation, had produced a report which, in completeness of detail and

as a whole, left nothing to be desired. The results achieved were of a nature to be of the greatest service in Public Health, and reflected high credit, alike on the efficiency of their sanitary authority in Edinburgh, and upon Dr H. Littlejohn personally. He felt he was only expressing the sense of the Society in desiring that the results of Dr H. Littlejohn's investigations should obtain the widest possible publicity.

Dr J. Bell rose to congratulate the Society on finding a second generation of Littlejohns rising up in their midst. He felt inclined to paraphrase the well-known lines of Horace into—

O patre acuto,
Filius acutior,

if only the lines would scan.

Dr James stated that at the late hour of the evening his remarks would be brief, and would take the form of inquiry rather than of criticism. He was of opinion that Dr Littlejohn had proved his case, and that the case of fever at the dairy-farm in the country was the effect of the same epidemic which had afterwards committed such havoc in Edinburgh. He held that the whole occurrence showed not only the advantage of the notification of diseases, but indicated the advisability of obtaining the notification of something more. For example, in this case, the milk was not the cause of the epidemic, it was the water which in one way or other got mixed with it, and, therefore, the indication was to look into or inspect the water-supply in dairies. He wished to know what could be done in this way at present. Then again legislative interference, if it is to be exercised with benefit to the community, must not be too hard on the individual, and Dr Littlejohn had told them that when the epidemic had been traced to this dairy, the milk-supply from it was stopped. As the milk itself was perfectly good, this was rather hard on the dairyman, and he wished to know what steps in such cases can be taken to prevent the loss, perhaps for days, of good milk and consequent loss to the dairyman.

Dr Harvey Littlejohn begged to thank the Society for their kind indulgence to his paper, and for the criticisms which had been offered on it. In regard to the exclusion of the water-supply and drains as causes of the epidemic, he thought that there could be no doubt, looking to the limited nature of the outbreak, that the water-supply had nothing to do with the origin of the disease, though, as stated in the paper, the deficiency in the water-supply at Rosemount Buildings may have influenced the progress of the cases. In the same way it is impossible, looking to all the circumstances of the outbreak, its simultaneous character, the one milk-supply, etc., to account for the outbreak by drain infection. It has been said that the evidence against the milk was not conclusive; except that no case of typhoid which would satis-

factorily account for the contamination of the well or milk was found at the farm, it certainly appeared to him impossible to avoid directly connecting the milk-supply from the farm with the outbreak in town. The other points raised in the discussion he thought had been fully treated of in the paper.

Meeting V.—February 4, 1891.

Professor A. R. Simpson, *President, in the Chair.*

I. Election of Ordinary Member.

The following gentleman was elected an Ordinary Member of the Society:—Dr Stewart Stirling, 6 Clifton Terrace.

II. Exhibition of Patient.

Dr John M. Balfour showed a patient, aged 25, in whom there was complete TRANSPOSITION OF THE VISCERA. After referring to the rarity of this condition, especially its recognition during life, the characteristic features were described. In the patient exhibited it could be determined that the heart, stomach, and spleen were situated on the right side, while the liver was on the left. The right testicle was lower than the left. The patient was, however, right handed, and the lateral curve of the spine in the dorsal region was to the right. No family tendency to malformation could be detected, and the patient himself was strong and healthy. Dr Balfour then gave a brief *résumé* of the theories advanced by Von Baer, Geoffroy St Hilaire, Rindfleisch, Virchow, Wehn, and Küchenmeister, in explanation of the condition.

III. Exhibition of Specimen.

Dr Harvey Littlejohn showed an ANEURISM OF THE HEART from a man, æt. 32, an old soldier, who had suddenly dropped down dead. No history of previous illness could be obtained. The heart was very much enlarged as seen *in situ* at the post-mortem, the enlargement being chiefly confined to the base. On opening the heart it was seen that this enlargement was not due to hypertrophy, but to a tumour of the septum. This tumour on examination was found to be an aneurism which opened into the aorta behind two of the aortic valves, and extended down into the septum of the heart for a distance of fully two inches. The cavity was full of loose white clot, and when extended was the size of a large hen's egg. The aortic valves showed great thickening, but in other respects the organ appeared to be healthy.

IV. Original Communications.

1. THE CARDIO-PNEUMATIC MOVEMENTS.

By John Berry Haycraft, M.D., D.Sc., F.R.S.E., and Robert Edie, M.B.

In addition to the chief movements of air, to and from the chest, which occur with every respiration, there are others of quite a minor character which seem to depend upon the circulatory system. These movements were discovered by Voit, and described and investigated by him, and more recently by Ceradini, Landois, and others. All these observers regarded them as due to *inflows* of air, to take the place of blood forced *from* the chest cavity, and *outflows* of air due to *inflows* of blood. As this explanation is not a correct one, it is unnecessary to go into the detail of their views, and we may give merely a short but sufficient sketch of them.

When the heart begins to contract, blood will be flowing into the chest as before, but the systolic pulse will not have reached the vessels at their outlets from the chest. The total result will therefore be that at this instant more blood will be within the chest than was the case before the commencement of systole, and in consequence of this air will leave the chest. As soon, however, as the chest is emptied by the systolic gush of blood, air will re-enter the cavity with a rush, and only leave it again when the diastolic inflow of blood has made itself felt. In addition to these, many other factors are believed by Landois to influence the movements, only one of which it is necessary to mention here. According to this observer, whenever the pulmonary arteries are distended by a systole they compress the air cavities, and cause an outflow of air from the mouth.

The cardio-pneumatic movements may be shown in a variety of ways, and one of the most striking is by means of the manometric flame. The mouth is brought in connexion with the capsule of a manometric flame, while the nostrils are closed and the glottis kept open, the tiny movements of air in the respiratory passages causing the flame to oscillate synchronously with the pulse or cardiac impulse. The lecturer demonstrated the movements to the members of the Society, and showed them also by means of a simple water manometer and a Marey's tambour.

He then proceeded to describe the experiments conducted by Dr Edie and by himself, showing in the first place pressure tracings they had obtained from the air cavities of the rabbit and sheep's lung, showing that during engorgement of the pulmonary arteries the air cavities of the bronchial tubes actually expand, the lung "behaving" like erectile tissue. Not only this portion of the theoretical explanation given by physiologists has to be given up, but the fundamental idea of the cause of the cardio-pneumatic move-

ments is erroneous, as could be demonstrated by a very simple experiment. They had taken a tracing of the cardio-pneumatic movements in the case of a rabbit, and then had opened the chest cavity and found, as they had anticipated, that the movements go on as before. These cannot, therefore, be produced in the way that Ceradini and Landois suppose, and another explanation must be forthcoming. It occurred to the experimenters that probably the chief cause of the cardio-pneumatic movements was the "massage" of the lungs by the heart during its contraction and relaxation. The heart alternately expands and contracts, pushing to and from the lungs which closely invest it. Whenever it expands it will press upon the lungs and cause an expiratory puff of air, and whenever it contracts an inspiratory puff will follow. To test the accuracy of this view they removed the anterior chest wall from a rabbit, somewhat inflated the lungs to prevent them collapsing and falling to the back of the chest, and took a cardio-pneumatic tracing. They then opened the pericardium and lifted up the heart away from the lungs by the aid of a pair of forceps, noticing that the movements almost entirely ceased, the tracing showing tiny oscillations, due probably to variation in pressure within the pulmonary arteries occurring with each cardiac systole. On allowing the heart once more to fall back upon the lungs, the tracing recommenced as before. Dr Edie and Dr Hayeraft, in pursuing their inquiry, took simultaneous tracings of the carotid and the cardio-pneumatic movements, and also of the apex beat and the cardio-pneumatic movements, demonstrating the method and the tracings to the Society. They were thus able to analyze their cardio-pneumatic tracings, and to find in them those parts which are formed simultaneously with the various events in the cardiac cycle. Beginning with systole, we find that there is a slight expiratory movement corresponding to the apex shock in the cardiograph, this is followed by a rapid inspiration as the heart contracts and assumes its smallest volume; as diastole occurs inspiration takes place once more. The cardio-pneumatic curve is, perhaps, the best indication we at present possess of the changes in size of the heart as a whole, for the lungs invest it and act as oncographs to the heart. The lowest part of the curve shows accurately the end of systole, a point difficult to determine on the cardiogram, and the apex shock is seen as well, and, moreover, is not exaggerated as it is in the cardiogram. It is much to be regretted that few people can keep their glottis open and hold the breath sufficiently steadily to enable good tracings to be taken. On this account cardio-pneumatic tracings can only occasionally be obtained from hospital patients. A full account of this research, and another upon the movements of the heart and the interpretation of the cardiogram, will be seen in the *Journal of Physiology*, vol. xi. pp. 486–495.

Dr Byrom Bramwell expressed the pleasure which he had derived from Dr Haycraft's admirable demonstration and lucid description of this important investigation. He thought Dr Haycraft was to be congratulated on the results which he had obtained; they at once seemed to commend themselves because of their simplicity. Dr Bramwell referred to the tracheal murmurs and tracheal pulsations which are met with in some cases of thoracic aneurism, and stated that these and some of the other physical signs met with in cardiac and respiratory diseases were doubtless produced in the manner which Dr Haycraft had described.

Dr Gibson was confident that by concurring in the laudatory remarks made by Dr Bramwell he was simply giving expression to the unanimous feeling of the Society. For his own part, in listening to the extremely able exposition of the cardio-pneumatic movements with which his friend Dr Haycraft had favoured the meeting, he had been greatly interested, not only in the facts mentioned, but in the explanations advanced in regard to a subject beset by considerable difficulty. In the days, now to be regarded as the dim and distant past, when he used to dabble in physiology, he had taken a good many cardio-pneumatic tracings, but not having had his interest stimulated in the matter, he had been content to accept the views of Landois, the only author whose writings on the subject he had read. He had listened to Dr Haycraft's explanation with much pleasure, inasmuch as it was at once marked by simplicity and lucidity, like most good working theories. He was in no mood to criticise the views that the Society had just heard, and would limit his remarks to two questions. Knowing the wide range of movement which the heart can exhibit on changing the position of any of the higher animals, he would, firstly, ask Dr Haycraft if the amplitude of the cardio-pneumatic movements underwent any alteration on placing the animals employed in the investigation in various positions? And, secondly, he would be much interested to know if in Dr Haycraft's opinion there was any means of determining the existence of a relation between these movements and the well-known wavy respiration?

Dr Smart, in congratulating Professor Haycraft on the extreme interest of his communication, remarked that, besides their scientific and physiological value, these cardio-pneumatic researches bore fruits of much clinical interest. To himself they were of especial interest, inasmuch as they corroborated in a striking manner certain views of his own in regard to the heart and respiration, which, without the proof of their correctness afforded by Voits, Ceradinis, Landois, and Haycraft's investigations, must have remained more or less speculative. One of the points in this connexion which he desired to bring under the notice of the Society, and which he thought deserving of special attention, was the occurrence under certain conditions of a blowing or puffing sound

connected, apparently, with the cardiac movements in their relation to the lungs. The conditions under which the sound occurs usually are—(1) exaggerated and intensified cardiac movements, (2) accelerated respiratory movements, (3) and a certain condition of the chest walls and internal organs favourable to conduction of sound, as exist, for example, in the production of "jerking respiration," which is a purely cardio-pneumatic phenomenon. The condition is predisposed to by cardiac hypertrophy and by whatever tends to diminish normal cardiac inhibition, *e.g.*, the use, in excess, of tobacco, alcohol, tea, etc. The puffing sound referred to occurs variously with the systole and diastole, and is sometimes audible with both, but more frequently he had noted its occurrence with the diastole. The puff bruit is usually associated with some bodily effort, as in running or other unwonted exertion or excitement which engenders cardiac agitation, and it is apt to be described as a latent bruit of valvular origin, or as an impurity of the first or second sound, implying the existence of lesion, thereby seriously prejudicing the health of the person immediately concerned. Besides other grave inconveniences, the detection of this sound is not the infrequent occasion of applicants for policies of life assurance being rejected on a mistaken diagnosis. Before the cardio-pneumatic researches were known to him, he had referred this sound to the cardiac movements in their co-relation to the lungs, and explained its origin as caused by the impact of the heart upon the inflated lung during systole and diastole, giving rise, in the former, to *ingress* of air in consequence of a slightly increased thoracic capacity by the systolic movement; and, in the latter, giving rise to *egress* of air by the sudden displacement of a portion of air from the inflated lung during diastole at the moment when the heart attains its maximum bulk and again falls back upon the lung. This rhythmical ingress and egress of the gases, but especially the latter, may, under the conditions referred to, become audible to the stethoscope as a bruit only of real diagnostic significance in so far as the physician is able to distinguish it from, and exclude, an organic lesion of the nature indicated. A second point of clinical import suggested by the cardio-pneumatic observations is, that the inspiratory and expiratory air currents induced by the cardiac movements virtually amount to a secondary, although a very subsidiary, mode of breathing, which, when the normal respiration from any cause is suspended, may suffice to aerate the lungs sufficiently as to maintain life for a time. It is doubtless owing to the air currents induced by the cardio-pneumatic movements that the blood of an hybernating animal is aerated, and its life maintained during its protracted repose. In proof of this he referred to the case of a patient in his ward whose breathing, in consequence of complete tetanus of the whole of the muscular respiratory system, including the diaphragm, was suspended for six hours, but whose heart the whole

time continued to beat vigorously at 56 per minute. Dr Smart considers it not improbable that this patient was kept alive during this period of respiratory suspension through the agency of the air currents established by the cardiac movements, which may not unwarrantably be viewed physiologically as a compensatory respiration. Dr Smart illustrated his explanation of the origin of the puff bruit by means of an inflated sack with a tube outlet. The rhythmical compression of the sack, by expelling the air through the tube, fairly represented the character of the puff bruit.

Dr Lundie wished to mention one fact in connexion with the subject of Dr Haycraft's admirable paper, viz., that he believed these movements could be demonstrated in healthy individuals to the ear as well as to the eye. It was many years since he heard the experiment tried; but while he was a student he used to hear it done thus: Sing a steady note very softly, and reduce the issuing current of air to as low a point as will continue to produce a sound. A rhythmical variation will then at least sometimes be heard like a 'beat,' synchronous with the heart movements. Such a method was, of course, of no value for accurate measurement.

2. A CASE OF HEREDITARY AMAUROSIS.

By William George Sym, M.D., F.R.C.S. Ed., Assistant Ophthalmic Surgeon, Royal Infirmary.

I was consulted recently by F. G., whose condition and history are as follows:—He is at present 36 years of age; is, and has always been in the enjoyment of perfect health, with the exception of occasional severe headaches. These headaches are usually chiefly occipital, and are regarded by him as "bilious;" they yield readily to medicines acting on the liver and bowels. They have troubled him ever since his school days.

In November 1879, being then 25 years of age, he was in Australia; and from being perfect, his sight degenerated within the space of a week to the state in which it is now. He tells me that on one Sunday he was able with ease to read off a small printed hymn-book in the hands of a person sitting in front of him; by the following Sunday he was blind. I mention these details in order to emphasize the rapidity with which the disease reached its maximum. His vision from the state in which it was at the end of that week has never altered for better or worse. On reflection, he thinks that perhaps his headaches were rather more frequent at that time than usual. He cannot be more emphatic in his statement than that. Except for this, he was in excellent health at the time. At that time he was a sailor, but employed temporarily on shore. His present condition as regards vision is that in the extreme temporal periphery of the fields he can perceive movements, and even, though with great difficulty, make out large black

letters on a white ground. Since the only available part of his field is so peripheral, he has, of course, no colour perception. He sees before him a light grayish mist or cloud, which appears to him to have the form, with either eye, of a triangle standing on its apex. It is particularly noteworthy that although this fog does not appear to him to be at all dense, yet I am doubtful whether he can through it perceive even the brightest light. He thinks he sees best in a dull light.

The pupils are equal and considerably dilated, and the media clear. The optic discs are perfectly white, but with a decided blue tinge; the arteries are much diminished in size, and the veins moderately so. There is perhaps a slight disturbance of pigment immediately round the discs, but elsewhere the fundus is perfectly normal; there are no special changes either in the macular region or at the periphery. The two discs are precisely alike.

Patient presents that form of nystagmus which is so often associated with bad vision coming on in the adult, viz., a slow, uncertain, irregular movement to one side, followed by a rapid flight to the other; in his case the slow movement is from left to right, the swift from right to left. This nystagmus renders very careful examination of the fundus well-nigh impossible.

He smokes, but not heavily; and before he lost his sight had only smoked for four years, and that very lightly. There is not the slightest ground for suspecting syphilis, either hereditary or acquired. The patient has always been strictly temperate, is not rheumatic nor gouty, nor is his family so; and is a strongly built, deep-chested, active, muscular, healthy-looking man. He has the fearless, bold style of walking which is met with in this form of disease,—so much so, that he finds it difficult to persuade the public that he is blind. This fearlessness, which is very extraordinary in a person who became rapidly blind at the age of 25, is at least partially explained by the fact that he retains peripheral vision, and is not uncommon in persons blind from this and similar diseases.

The patient's family history is very striking:—His father, a hale old man, is alive at the age of 77, in the enjoyment of good sight and good health. His mother is alive, well, and *blind* at the age of 75, having lost her sight at the age of 51. She has always been subject to somewhat severe headaches.

My patient is one of a family of five, accounted for thus:—

1. Daughter, æt. 51. In perfect health, with perfect sight. She is married, and has two sons and two daughters, the eldest of her family being 18, the youngest 12; all see well.

2. Son, æt. 47. A large, powerful man, in perfect health, but *blind* from his twentieth year. Like my patient, he was a sailor, and the only serious disease from which he has suffered is yellow fever, which he had at Havannah. It was a very severe outbreak, and out of a ship's company of twenty-one, he was the only person

left alive. According to his medical attendant (so I am told) he owes his life, according to his friends he owes his blindness, to the vigorous cupping to which he was subjected in the treatment of the fever. It was very shortly after this that he became blind. He has never suffered from headaches.

3. Son, æt. 39. Also a strong, healthy man, but *blind*, he having become so four years ago. Neither has he been a sufferer from headaches.

4. Son, my patient. He has two sons, aged 8 and 6, both in good health and both seeing well.

5. Daughter, æt. 32. She is quite well and sees perfectly. She is married and has a young family, all the members of which see well.

My patient knows little of his grandparents or of his uncles and aunts, but believes that there have been no cases of blindness among them. Among his cousins, who are numerous, the only case is that of a son of his mother's sister, who became blind at the age of 26, the alleged cause being a fall on the ice on the back of his head. Whether this be the real cause or no is not certain, but we must let the statement pass for what it is worth.

As regards the blindness of the patient's mother and brothers, I have only to say that they were attacked in precisely the same way as my patient, and that their state of vision is much the same as his, but not quite so bad, and blindness took rather longer to reach its maximum. They are all hemeralopic, all complain of a grayish cloud in the centre of the field, all became blind rapidly, and all retain their peripheral vision.

Here, then, we have an account of a family in which the mother and three sons are all blind, while the father and two daughters have escaped, all being in good health.

The disease which has shown itself in this family, and has exhibited so much care and discrimination in the selection of its victims, is fortunately rare, but is by no means unknown. I will not trouble you with quotations from the older authors on eye disease, who give analogous family histories, because one never can be sure of one's ground when speaking of cases of "Amaurosis" in the pre-ophthalmoscopic days. The most exhaustive account of the affection yet published is that from the hand of Leber in the *Graefe-Saemisch Handbuch*, which was based upon an unusually large experience of the disease—fifty-five cases in sixteen families. Interesting notes and papers on the disease are to be found in Landolt and de Wecker's *Traité Complet d'Ophthalmologie* (de Wecker);[1] in *Graefe's Archiv.* (Leber);[2] in *Zehender's Klinische Monatsblätter* (Fuchs);[3] in *Medical Times and Gazette*, 1862 (Sedgewick); and in *Ophthalmic Review*, 1885 (Story).[4]

[1] Vol. iv. [2] Vols. xvii., ii. [3] 1879, p. 332.

[4] Also, I believe, in the *Berlin. Klin. Wochenschrift*, 1876 (Pufahl), and in *Trans. Amer. Ophthal. Soc.*, 1884 (Norris), but upon these papers I have not been able to lay hands.

The disease, although called hereditary, has not so much tendency to descend as to show itself in several members of the same family, while the hereditary liability, the *Erblichkeit*, seems to be comparatively slight. In only one instance with which I am acquainted, recorded by Lucas and quoted by Leber, did the disease appear in three generations,—in at least two generations affecting women, curiously enough. Women, as is the case with optic nerve diseases generally, and certain other diseases also, are much less often affected than men (the proportion is about one to ten).[1] When women are attacked it is seldom those who have borne children. Very rarely has it been found that a blind mother, or a mother who has subsequently become blind, has borne sons who have also been thus affected. The healthy sisters of blind brothers have more often borne sons who became diseased. These points are well exemplified in a family known to Fuchs (Arlt's Klinik). This family consisted of two sons and four daughters. The two sons became blind; one was married, but all his family saw well. The daughters all saw well; one of them married a healthy man, and had a family of five sons and one daughter. Most, probably all, her sons were affected; the daughter and her family escaped. Such family histories remind one of those of the hæmorrhagic diathesis.

The usual period of life at which victims are affected is from 18 to 28 years (de Wecker); but when women are affected, the menopause is not unfrequently the time selected. But the disease has been observed as early as the fifth year and in men above the age of 40. The onset of the disease is usually rapid, sometimes very rapid, and apparently quite unprovoked; within a very few weeks or even days, a patient, who is all the while in good health, loses his sight. After this time some improvement may take place, sometimes even great improvement, as in one or two of Leber's cases; but more frequently a further slow deterioration goes on for months. Absolute recovery is unknown, although some few have come near to it, and absolute blindness is very rare. The final result in my patient's case appears to be considerably worse than the usual termination. The symptom of which the patient most frequently complains is simply rapidly advancing blindness; but often there is mention of a feeling as of a cloud or mist before the eye, which cloud does not appear dense, although over its area there may be absolute blindness. In all cases there is a central scotoma, more or less large and more or less absolute, while the periphery is almost invariably unaffected. Leber observes that although colour vision is always damaged eventually, it is sometimes normal at first; while peripheral vision may be lost at first, though it is always recovered later, and that usually perfectly. Hemeralopia is a very common symptom. Both eyes are invariably affected, although one may precede the other by a short time,

[1] See *Ophthalmic Review*, January 1891.

and one may be more severely damaged than the other. Occasionally, but rarely, there are photopsiæ, pain on movement of the globe, etc. A good number of the patients have suffered for years from headache. In a number of the families in which the disease has appeared, it has been noticed that mental or nervous diseases were also common, affecting both those who were blind and those who were not,—particularly notable in this relation are the cases of Sedgewick and Story. I could find no such history in my case. In only one case has consanguinity of the ancestors been found, and then it was that the patient's great-grandparents had been related!

The ophthalmoscopic appearances are in the very early stages unchanged from the normal; then appears a congestion of the discs; the veins full and somewhat tortuous; the margins of the disc blurred. In some cases there has been a decided neuro-retinitis. About the time that the rapid deterioration of vision ceases, this gives way to an increasing pallor of the disc, along with diminution in size of the vessels, particularly of the arteries. This white or blue-white pallor is always most marked in the outer quadrant of the disc; sometimes it has been seen there only, the region in which the papillo-macular bundle of fibres is believed to run. This process of decolorizing of the disc goes on whether the vision be slowly improving or slowly diminishing, and appears to be perfectly unaffected by treatment.

In the way of treatment various drugs have been used, chief among these being mercury and strychnine; in the early stages local depletion is recommended; galvanism was at one time believed to be going to be useful, and in at least one case of Leber's did seem to produce great benefit; but the good effect was not lasting, and in other cases it has failed.

In regard to the pathology of the condition, there seems little doubt that it is a retro-bulbar neuritis. To this conclusion we are pointed by its rapid onset, the presence of a central scotoma, particularly for colours, with retention of good peripheral vision, the hemeralopia, and the fundal changes, which appear at first sight to be inconsistent with the alterations in subjective symptoms, though, as we now know, they are not really so. Believing the condition to be a neuritis, I prefer to name the disease Hereditary Amaurosis rather than Hereditary Optic Atrophy; for in this disease the atrophy is secondary to the lesion which causes the blindness; and also, and more particularly, because true optic atrophy, with quite different symptoms, has been found as a hereditary condition.

Note.—In this paper I have used the word Hemeralopia in what I believe to be its true signification, viz., that condition in which sight is bad in a bright light, and improves in a dull light; Nyctalopia indicating a precisely opposite state of vision. Until all observers are agreed upon the meanings of these two words, it

is better that each one using them should define his intention in their use, since some oculists mean by hemeralopia exactly that condition which some others employ nyctalopia to express.

Dr Lundie had seen several cases of the form of disease described by Dr Sym; but all in the same stationary condition as his, with absolute blindness at the centre of the field, and some vision remaining at the periphery. It would be of special interest to know whether Dr Sym's patient and his brothers had families; for on the analogy of some similar family diseases in which crossed heredity occurs, it might be expected that their daughters would be likely to become affected.

Dr Sym emphasized the fact that in his and numerous other similar cases no explanation of the occurrence of the disease could be found. There seemed to be no family diathesis pointing to gout or other disease so far as he had been able to make out. He recommended to the notice of members of the Society who were interested in the matter the paper by Jensen on Central Scotomata, translated in the *Ophthalmic Review* of January 1891. The disease is referred to in that article under the name "Stationary Scotomatous Optic Atrophy."

Meeting VI.—March 3, 1891.

Professor A. R. Simpson, *President, in the Chair.*

I. Election of Ordinary Member.

The following gentleman was elected an Ordinary Member of the Society:—Francis D. Boyd, M.B., C.M., 3 Coates Place.

II. Exhibition of Patient.

Dr Byrom Bramwell exhibited a case of Freidreich's ataxia. The patient, a boy, æt. 14, first came under his observation some three years ago, complaining of difficulty of walking, the result of inco-ordination and not of loss of muscular power; the knee-jerks were at that time abolished. During the past three years the disease had steadily and somewhat rapidly increased, and the patient now presented all the characteristic symptoms of the disease—in fact the case was a typical one. No other member of the patient's family was affected.

III. Exhibition of Specimens.

1. *Dr Byrom Bramwell* exhibited—(1.) Two Cases of brain tumour.

The *first*—a glioma of the cerebellum—was of great interest from the fact that under large doses of iodide of potassium all the symptoms (with the exception of optic atrophy) had for a time almost completely disappeared. Dr Bramwell had had the patient under observation for some years; the symptoms were typical and characteristic of cerebellar disease, and the diagnosis, as the photographs showed, was confirmed on post-mortem examination. The *second*—a large tumour in the region of the pituitary body—was of great interest, for it showed the value of determining the condition of the fields of vision in cases of localized cerebral disease. From the character and form of the hemianopsia which present, the diagnosis of a tumour pressing upon the chiasma had been confidently made. (2.) Photograph of case of FACIAL HEMIATROPHY. The patient, a young lady, had suffered from the condition for some four years; the appearances were highly characteristic, the left side of the face being markedly atrophied, and in places hard and sclerodermatous. The left side of the tongue was markedly wasted; this was a point of great interest, for it showed either that the fifth nerve possessed some nutritive influence upon the muscular fibres of the tongue, or that the lesion which commenced in the trophic centres connected with the fifth nerve had extended (flowed over) to the trophic centres connected with the hypoglossal. Dr Bramwell contrasted the symptoms of the present case with those of another one which he had published about a year ago.

2. *Dr Affleck* showed a case of VASO-MOTOR DISTURBANCE OF THE HANDS, causing blueness or lividity of the skin, which had existed for three months. The lividity was symmetrical. The patient, a young woman of 20, was quite healthy in every other respect. There was a history of injury to the left hand and arm three years ago, and pain in that hand ever since. There is complete cutaneous anæsthesia of the ulnar side of left hand and portion of forearm. Both hands, besides being deeply livid, are almost constantly cold. The case resembles the "local asphyxia" stage of Raynaud's disease; and although not entirely conforming to that condition, appears to have more affinity with it than with any other known ailment. It is not an example of the "blue œdema of the hysterical" described by Charcot, which is unilateral. A point of interest in reference to the pathology of the complaint is that while a chronic peripheral neuritis due to injury might have accounted for the disturbed condition of the circulation in the left hand, it could not account directly for the lividity of the right hand in which there was no evidence of neuritis. These vaso-motor neuroses were deserving of more attention, particularly with a view to classification, than they had yet received.

3. *Dr W. Russell* showed a LUNG WITH MULTIPLE SARCOMATOUS TUMOURS, taken from a young woman from whom a large sarco-

matous tumour had been removed by Mr Duncan from the left thigh. There were no growths in any organ save the lungs.

4. *Mr Caird* exhibited a preparation and drawing from a case of INFANTILE HERNIA. The patient suffered from an irreducible inguinal hernia, complicated with a hydrocele. This having been emptied, the patient desired a radical cure for the removal of a firm mass which lay in the canal. On operation there was found a large tunica vaginalis into which the hernial sac projected. This was opened, and was found to contain a large mass of felted omentum.

IV. Original Communications.

1. STRICTURE OF THE ŒSOPHAGUS—GASTROSTOMY.

By John Duncan, LL.D., *F.*R.C.S. Ed., Senior Surgeon, Royal Infirmary; Lecturer on Clinical Surgery, Edinburgh School of Medicine.

I PURPOSE in this paper directing attention to the operation of gastrostomy as performed for stricture of the œsophagus.

My last case was that of a man of 45, who, when admitted to the Infirmary, had been unable to swallow anything except a little liquid for several days. He had experienced difficulty in swallowing for three months, and was very greatly emaciated. Squamous-celled epithelioma was diagnosed. A few days were spent in endeavouring to improve his condition by rectal feeding, and then gastrostomy was performed. He left the Infirmary a month afterwards, when he was taking large meals by the tube, and had gained nearly a stone in weight. After three months he died at home from extension of the disease to surrounding parts in the thorax.

This is a very typical case of malignant stricture of the œsophagus, and illustrates well the points to which I wish to direct attention.

Naturally the first question with which the surgeon concerns himself in these cases is that of diagnosis. How does one come to the conclusion that such as this is a squamous-celled epithelioma?

In order to decide the question, it is necessary to call to mind the various causes of œsophageal obstruction, and to inquire how they fit into the symptoms exhibited by the patient. Now dysphagia may be produced by extrinsic pressure or by intrinsic disease. We therefore eliminate first, by most careful examination, all possible sources of the former, such as goitre or aneurism, or other swelling, whether of an inflammatory or neoplastic character. So limited, our search is then directed to affections of the œsophagus properly so called.

Stricture may arise in the œsophagus from spasm, inflammation, syphilis, benign tumour, traumatism, and malignant disease—

1. Spasmodic stricture is intermittent, sudden, accompanied by some form of dyspepsia, or associated with neurotic disturbance. It is necessary to bear in mind that as in other strictured canals

there is sometimes an element of spasm in organic disease. This for a time may obscure the diagnosis, and, at all events, it is necessary in every case to obtain all the information which can be got from the passage of bougies. There are one or two points in connexion with this little operation to which it may be well, therefore, to direct attention.

Especially in neurotic patients, but to a greater or less extent in all, difficulty may be experienced in entering the œsophagus. This may be overcome by directing the patient to swallow at the moment when the bougie reaches the orifice of the gullet. Now the movements of swallowing are normally preceded by closure of the mouth and pressing of the tongue against the palate, and to enable the patient to swallow with open mouth he must receive the assistance of the surgeon. The head should be thrown back, and the surgeon's forefinger should be used to fix the jaw and afford a fulcrum especially to the anterior part of the tongue. The muscles which elevate the larynx thus gain a fixed point, and when brought into action cause the air passage to glide upwards and forwards over the bougie.

I have mentioned this point merely as an incident, and not as likely to lead to error in diagnosis. I have seen the error made, but there is little practical difficulty in distinguishing between this form of obstruction and stricture. The latter, in fact, very rarely occurs in this situation unless by extension from the air passage, and is within reach of the finger when it does.

This little difficulty overcome, the bougie normally glides easily into the stomach. I have, however, known doubt in determining whether the bougie has reached the stomach or not, and this matter is important, because not infrequently the stricture is situated at the very end of the œsophagus. On entering the stomach, the sensation of having reached a cavity is experienced, the bougie passes onwards with a diminished resistance and to a length which cannot be accounted for by supposing that the diseased part is pushed on in front of it.

This last must indeed be accompanied by a feeling of increased resistance; and I believe it may be said that if you are in doubt as to whether or not the bougie has reached the stomach, you may take it for granted that it has not reached it.

Resistance, then, implies abnormality, but very little further. You cannot even thereby determine the presence or absence of a diverticulum. My own experience of spasmodic stricture is that a bougie of moderate size can always be passed with gentle pressure. I have been rather astonished in these cases by the absence of any grasping of the instrument after it has passed or on withdrawal. Such grasping is very marked in traumatic stricture, but not in spasmodic.

2. Having eliminated the spastic stricture, we have next to consider the possibility of the obstruction being inflammatory. So

far I have seen this condition only in the alcoholic. Two cases have come under my observation in which habitual drunkards became afflicted with extreme dysphagia. One died of cirrhosis of the liver, the other of delirium tremens. In both, efforts to pass a tube for purposes of alimentation had been repeatedly and unsuccessfully made, and it was thought that organic stricture existed. On post-mortem examination, it was found (I was present at the sectio in only one case) that the lumen of the œsophagus was not diminished, but that throughout its length its wall was much thickened and œdematous, and its mucous membrane covered by many small ulcerations. The difficulty in passing an instrument had been due to the absence of pliability and of peristaltic action. You will find it difficult to pass an instrument into the stomach on the cadaver. The absence, then, of any history of alcoholism and of the pain which was present —especially on trying to pass an instrument—in these cases I regard as valuable points in their diagnosis.

3. Syphilis, in its tertiary form, may attack the œsophagus as it may any portion of the body, and plainly, if so, may cause dysphagia either as a gumma or cicatrix. But it is very rare. I remember only one case. In it the history and concomitants of the disease were very clear, and the man recovered under mercury, iodide of potassium, and the use of the bougie.

4. Benign tumours of the œsophagus (polypoid generally) are also exceedingly rare, and do not usually give rise to dysphagia. Most commonly they have not been diagnosed during life.

5. Traumatic stricture arises from swallowing caustic substances, or possibly from wounds, it being, of course, essential that the traumatism of whatever kind be circumferential in extension. The history should therefore enable us to eliminate this form. I have met with one example of concealment of the cause due to the fact that the swallowing of the caustic was with suicidal intent, and it is not impossible that intoxication may interfere with the reliability of the information. But, as a rule, the history, as given either by the patient or his friends, is clear and decisive.

You will see, then, that we arrive at the diagnosis of malignant stricture largely by a process of exclusion. One may almost safely say that, if a stricture of the œsophagus be idiopathic, gradually increasing, and of comparatively recent origin, it is malignant. Blood and discharge, and microscopic evidence of destruction of tissue in the matter vomited, may confirm the diagnosis.

Of malignant tumours of the œsophagus, by far the largest number are squamous-celled carcinomata. Sarcoma as a primary affection is almost unknown. It may occur by extension of the disease from neighbouring parts, and I was once present at a sectio in which we found an œsophageal stricture, small, annular, and tight, in a patient who had been repeatedly operated on during

a period of thirty years for what was then called "recurrent fibroid" of the thoracic wall. I have at present under my care a case of stricture of the œsophagus, sent me by Dr E. Carmichael, in whom there is also an undoubted sarcoma of the scapula. The immense probability is, then, that if malignant, we have to deal with a case of epithelioma.

The diagnosis established, we turn next to the question of treatment, and the first proposition which I would submit is, that if the disease be incapable of free removal (and most are so), the first consideration is to put it at rest, and that local interference is only to be justified when it is the lesser evil. I have not had an opportunity of removing a malignant affection of the œsophagus. It has been done once or twice with not very fortunate result, but I believe the operation to be justifiable if the tumour be within reach.

But while we interfere with the growth as little as may be, it is plain that we cannot allow the patient to die of starvation; some means of alimentation must be employed. There are several. First, there is rectal feeding, and much may thus be done. By in this way giving the œsophagus a complete rest it is not infrequently found that temporarily the passage becomes again patent, and by mouth and rectum the patient may be fairly sustained. But if we be reduced to feeding by the rectum only, the patient starves slowly, but still surely, while in certain cases the rectum proves refractory to nourishment.

Attempts have therefore been made to dilate or divide the stricture, or to lodge a tube in its lumen. In those, especially if they be aged, in whom the occasional passage of a soft tube is easy, maintains the patency, and affords a fair opportunity for nourishment, life will probably be prolonged as much as by any other means. But, on the other hand, it has been abundantly proved that the discomforts, disadvantages, and dangers of internal division, permanent lodgments, or frequent passages of instruments, simply hasten the inevitable end.

The operation of œsophagostomy, or opening the œsophagus below the strictured point, is plainly applicable to only a limited number of cases. Malignant stricture is usually too low in the tube. There are, moreover, certain disadvantages inherent in the operation. It involves the possibility of being ineffective, because there is a second stricture on a lower level, and it possesses a high rate of mortality.

But we have yet another method at command—the operation of gastrostomy—which I desire to commend to your consideration. Some surgeons have recently condemned the operation, as I think somewhat hastily, for I feel sure that if cases be properly selected and the operation carefully done, it greatly prolongs life, and adds enormously to the comfort of the patient. There can be no doubt that gastrostomy cannot be performed without liability to a con-

siderable amount of shock. The nervous arrangements of these parts, it has long been recognised, react strongly upon injury, and the patients on whom gastrostomy is performed are necessarily of advanced age and already enfeebled by imperfect alimentation. I performed it in a man of 75, hale for his years, but who had, before entering the Infirmary, been almost absolutely without food for a week. We endeavoured for some days to bring up his strength by feeding him per rectum, but without much effect, and rather than see him die of starvation I operated. His temperature and pulse fell quickly, and I opened the stomach next day and fed him thereby. He did not rally, and died from cardiac collapse fifty-four hours after the operation. I should hesitate, certainly, to refuse gastrostomy on account of exhaustion unless it were very extreme. There are people old and apparently worn-out who yet suffer little from what we call shock. I have successfully operated in a man not certainly so old as the one I have mentioned, but older in appearance and more emaciated. In him there was no shock whatever. But I should recommend that the operation be not too long delayed. When the passage has become so far closed that it is impossible to administer sufficient food to supply the natural waste of the body, the stomach should be opened. Shock is really the only unavoidable danger. The mortality which has attended the operation from other causes our modern surgery is capable of overcoming,—the results, I mean, of septicity, whether local or general.

It is of immense importance, then, that the operation should be so performed that the patient shall be carried through the inevitable shock, and, of course, protected at the same time against septic infection of the peritoneum.

An incision is made in the wall of the abdomen, with the usual precautions, preferably parallel to the rib edge. I then pass my finger along the viscus, which I take to be the stomach, until I ascertain that it is so by feeling its passage through the diaphragm. In some cases you are then able also to feel the disease, and from its size to form a probable forecast of its future progress. I then fix the stomach to the wall of the abdomen by four encircling sutures, which penetrate the whole thickness of that wall, and by many radiating, which penetrate only skin and peritoneum. In both cases the stitches include the serous and muscular coats of the stomach. I then make a small opening in the stomach and introduce a soft rubber tube of the size of No. 8 catheter. This immediate opening I believe to be of great moment. At first I allowed several days to elapse before opening the stomach, but I have gradually shortened the period, until now I open at once, and proceed immediately with small but frequent doses of liquid food. In order to diminish, as much as may be, the chance of septic infection of the peritoneum, I take care that the tube shall closely fit the opening in the stomach, I secure its orifice by a clamp, and

surround it with powdered boracic acid. There is, in truth, no tendency to the egress of fluid from the stomach, and in no case have we seen the slightest inflammatory reaction. The result has been most satisfactory in lessening the amount of shock. There is yet in most cases a slight depression of pulse and temperature for two or three days, but immediately thereafter the patient rallies, and begins to increase in strength and put on flesh.

Under these circumstances the only point that remains is to determine the advantage which the patient may derive from the operation.

I have had two deaths from it. To one I have already referred, in whom the exhaustion before and the collapse after operation were so great that I was induced to open the stomach the next day. I believe that this farther interference rather increased the shock, but it proved to me the safety of early incision, and led to the adoption of immediate opening, which has at once the effect of diminishing the chance of shock from manipulation, and allowing nourishment and stimulation to be immediately administered.

The other death was that of a man æt. 57, in whom I delayed opening till the fifth day. He was excessively weak and emaciated at the time, and I believe the second interference was injurious, and was certainly too long postponed. He died on the sixth day. There was no peritonitis. He died simply of exhaustion; and at the post-mortem examination the disease was found to be very extensive, and to have reached down so far as to occupy a considerable area of the cardiac end of the stomach.

My other cases have been five in number, and they lived respectively for eleven, ten, five, four, and three months after operation. In every case the patient rapidly increased in weight during his residence in the Infirmary. One, a patient of Dr Pope, travelled from South Shields five months afterwards simply to show himself. He had been able, after leaving the Infirmary, to feed largely on pappy substances by the mouth, as the rest which the operation had given to the œsophagus had, as often happens, allowed the swelling somewhat to subside and the lumen to expand. All died ultimately of extension of the disease except one, a patient of Dr Robert Moir, who succumbed, I believe, to an intercurrent attack of bronchitis.

When it is considered that in none of these seven had the patient more than a few weeks of life before him had he been left alone, that the two who died were on the extremest verge of absolute starvation, that those who lived had in every case months of comfort, and in some of apparently perfect health, and that in them one effect undoubtedly was to diminish by rest the rapidity with which the disease was spreading,—when these things are considered, it is difficult to avoid the conclusion that, when the patient has

begun to suffer from insufficient nutrition, the operation ought to be performed, and that it need not even be denied when the marasmus is far advanced.

If, again, the operation be justifiable in malignant disease as a euthanasia, *a fortiori* it is to be advised in traumatic or cicatricial stricture. Whether or not Loreta's method of stretching be combined with it, it holds out to such patients a quite indefinite prospect of longevity. I have not had an opportunity of performing it myself. Only three such cases have ever come under my care. One of them was the syphilitic gumma already referred to, which I think was more largely inflammatory than cicatricial. The others, a man and woman, were both successfully treated by dilatation. The man had a stricture, the result of swallowing caustic potash solution, so tight that only a bougie of urethral size No. 4 could be passed. With immense patience it was ultimately overcome, and he left the Infirmary able to pass a large-sized bougie for himself. Parenthetically I may observe that I found in his case that the only way to advance in the early stage of his treatment was by the daily passage of the instrument.

I have been able, then, to avoid operation in traumatic stricture, but I have no hesitation in saying that, in cases which resist dilatation (and dilatation means an infinite amount of delicacy and care), gastrostomy ought to be performed.

The President expressed the pleasure with which they had all listened to Mr Duncan's account of his experience in the performance of gastrostomy, and was sure that they anticipated with delight the prospect of having further communications from him in relation to the surgery of the abdomen. As one who had frequent occasion to open the abdomen for pelvic disease, he (the President) had been specially interested in hearing what such a master in surgery as Mr Duncan had to say of the conditions of safety in opening into the abdomen in its higher planes. He thought Mr Duncan had arrived at a correct conclusion in deciding that, where the stomach had to be opened, it was better to open it at the time that the incision was made through the abdominal wall. First to stitch the stomach to the abdominal parietes, and at a later date to cut through the stomach wall was simply to subject the patient to a double risk, and retard the benefit which could be obtained by the immediate introduction of aliment into the system.

Mr Caird joined the President in thanking Dr Duncan for his paper, which should do much to rescue gastrostomy from the position which it was so apt to occupy with its former high mortality. He thought œsophagostomy need not be so uniformly fatal as represented, and referred to a successful case shown by Mr Chiene a few years ago. He believed that much of the shock

was due to the general anæsthetic, and had himself attempted the operation under cocaine. At the conclusion of the operation the patient exhibited less collapse and shock than he had ever before seen after a gastrostomy, but the patient ultimately sank.

Prof. Greenfield, under whose care the patient mentioned by Mr Caird had been, could not concur in the view that the case was in favour of the avoidance of a general anæsthetic. The risks of shock in such operations were great; but unless some more perfect local anæsthetic were found, the strain upon the patient was probably greater than if a general anæsthetic were given.

Dr Troup mentioned that twenty-three years ago he performed gastrostomy, and opened the stomach at once and commenced to feed the patient. Dr Troup had only seen gastrostomy performed once since then, and told the operator that he made too much ceremony, and should open at once, and Dr Troup was glad that Dr Duncan approved of the immediate opening. Dr Troup's case is related in the *Edinburgh Medical Journal* of 1872, and was the first in Scotland.

Dr W. Russell thanked Mr Duncan for the information in his paper. As a pathologist it seemed to him that the mortality in cases of abdominal section for various diseased conditions was great, and he was glad to have the other side presented, for, as a physician, the question to him was whether it was an operation to be recommended to one's patients. Statistics of Mr Duncan's kind would do much to settle the advisability of recommending operation in various abdominal conditions.

Mr Duncan said that he was by no means prepared to hold that the comparative value of the various operations for alimentation in œsophageal stricture was finally decided. Prof. Gross, however, had shown statistically that the mortality of œsophagostomy was distinctly greater than that of gastrostomy, and it was evident that the largest cause of mortality in the latter, the septic, could be obviated by modern methods, while it must necessarily remain in the former. Moreover, œsophagostomy was applicable to comparatively few cases—to not one of his own, for example. On theoretical grounds, and from examination of published reports, he must provisionally come to the conclusion that the lodgment of a permanent tube was not likely to become an established mode of procedure. While quite admitting that the anæsthetic added temporarily to the shock, he was not inclined to think its effect on the mortality was large, but to attribute it chiefly to the age and exhaustion of the patient.

2. THE SYMPTOMS OF MYXŒDEMA AND EXOPHTHALMIC GOITRE CONTRASTED.

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The clinical features of myxœdema are, in many respects, the direct opposite of those of exophthalmic goitre.

The two diseases resemble one another, inasmuch as that in both the thyroid gland is diseased—atrophied in myxœdema, and (usually) hypertrophied in exophthalmic goitre—and inasmuch as both diseases affect women much more frequently than men.

But in their mode of development and the character of many of their symptoms the two diseases are very different.

The symptoms of myxœdema are almost always developed in a very slow, insidious, and gradual manner;[1] the symptoms of exophthalmic goitre not unfrequently develop with considerable rapidity, sometimes, indeed, suddenly after a fright or other profound emotional disturbance. The age at which myxœdema is developed is, as a rule, much later than that at which exophthalmic goitre occurs. The ordinary adult form of myxœdema usually develops after the age of 30; the disease is very rarely indeed developed between the age of infancy and 30, *i.e.*, during early adult life (15 to 25). Exophthalmic goitre, on the contrary, very often, indeed in fact usually, develops between the ages of 15 and 25. Myxœdema is most frequent in married women who have borne large families; while exophthalmic goitre is most common in unmarried (single) women, and (?) in married women who have not borne children. Pregnancy seems, as a rule, to exert a prejudicial influence on patients who are affected with myxœdema; while, in some cases of exophthalmic goitre, it seems to prove beneficial or even curative. The thyroid gland is atrophied and degenerated, and its functions diminished or abolished in myxœdema; while in exophthalmic goitre the thyroid gland is (usually) hypertrophied and its functions increased and perhaps perverted in character. In myxœdema the temperature is subnormal, and the patient feels the cold; while in exophthalmic goitre temporary elevations of temperature are apt to occur, and the patient feels the heat and complains of flushings. In myxœdema the skin is always dry and harsh and the secretion of sweat abolished or greatly diminished, while in exophthalmic goitre the skin is soft and moist, and profuse sweating is often a prominent symptom. In myxœdema the electrical resistance of the skin is greatly

[1] A few cases have been reported in which the symptoms of myxœdema are said to have developed more or less rapidly, but in such cases the disease had probably been in existence for some time unknown to the patient.

increased, while in exophthalmic goitre it is greatly diminished.[1] In myxœdema the bowels are usually constipated; whereas, in exophthalmic goitre, attacks of diarrhœa are apt to occur. In myxœdema menstruation is usually arrested, while in exophthalmic goitre menorrhagia is generally (according to one eminent gynæcological friend, always) present. In myxœdema, placidity, diminished emotional activity, and slowness of all nerve processes, are conspicuous features; while in exophthalmic goitre, emotional excitability, irritability, and nervousness are always very marked.

The contrast which the two diseases present is very interesting when it is remembered that in myxœdema the thyroid gland is degenerated and atrophied, while in exophthalmic goitre it is (usually) hypertrophied and enlarged.

There is every reason to suppose that the clinical condition, which we term myxœdema, is the direct result of abolition of the function of the thyroid gland. Whether the atrophy and degeneration of the thyroid gland is primary or secondary is, however, another matter. Victor Horsley has suggested that the degeneration of the thyroid gland is, perhaps, secondary to some change, at present unknown, in the nervous system—that it is, in short, secondary to some unknown primary nerve lesion. Possibly this may be so, but as yet there are, so far as I know, no definite facts to support such a view. But be that as it may, almost all observers are agreed in thinking that the nutrition of the nerve centres, and especially the higher cerebral centres, is in some way or another dependent upon the healthy functional activity of the thyroid gland; and that when the functional activity of the thyroid gland is abolished or diminished in consequence of degeneration and atrophy, the nutrition of the brain suffers—in short, that the nervous and other symptoms, which go to form the clinical picture of myxœdema, are due to abolition of the function of the thyroid gland.

Now, accepting this view, and bearing in mind, *firstly*, that in exophthalmic goitre many of the symptoms are the direct opposite of those of myxœdema, and, *secondly*, that in many cases of exophthalmic goitre the thyroid gland is hypertrophied and its function presumably in excess, one is tempted to theorize that the symptoms of exophthalmic goitre, or, at all events, those symptoms

[1] The difference in the electrical resistance of the skin in the two diseases is no doubt due to the different condition of the skin as regards moisture in the two diseases. I entirely agree with Dr H. W. D. Cardew (see *Lancet*, February 28, 1891), that the diminished electrical resistance in the skin which is such a striking feature in most cases of exophthalmic goitre is due to the fact that in Graves' disease the skin is usually steeped in moisture. Dr Cardew says (p. 484), "Most patients suffering from Graves' disease perspire more or less profusely, and as evaporation does not take place at a rate equal to that of secretion, the stratum corneum is covered with sensible perspiration, and its intercellular interstices and the orifices piercing it are filled with saline solution, which forms an excellent conducting medium between the internal fluid of the body and the electrodes placed on it."

which are the direct opposite of the symptoms of myxœdema, are the result of excessive functional activity of the thyroid gland.

Such a view is, to my mind, very plausible. The main argument which can be brought against it, viz., that the thyroid gland is not perceptibly enlarged in some cases of Graves' disease in which nerve symptoms are very prominent, is not perhaps absolutely conclusive. It may, perhaps, be theorized that, in such cases, the functional activity of the gland is deranged, and that in consequence of the perverted secretion of the gland the nutrition of the nerve centres is impaired, and the emotional excitability and other symptoms, which are the reverse of those of myxœdema, are produced.

The whole subject of the pathology of exophthalmic goitre is at present very obscure. Even if the theory which has just been advanced should ultimately prove to have some more substantial basis than the contrast which the symptomatology of myxœdema and exophthalmic goitre suggests, the ultimate pathology—the primary cause of exophthalmic goitre—remains very much in its present position; for, so far as our present knowledge enables us to judge, the altered functional activity of the thyroid gland (whether it be a simple increase the result of hypertrophy of the organ, or a perversion of function) is only a part of the condition. The increased or perverted functional activity of the gland may perhaps be the cause of some of the symptoms—those which contrast so remarkably with the symptoms of myxœdema—but, so far as our present knowledge enables us to judge, it seems hardly able to account for the increased frequency of the cardiac action, the exophthalmos, and some of the other eye symptoms (Von Graefe's sign and Stellwag's symptom, for example), which are usually such striking features of the disease. Certainly, so far as my observation enables me to judge, the reverse of these conditions—diminished frequency of the heart's action and retraction of the eyeballs—are not present in myxœdema.

It seems probable, therefore, that the primary cause of Graves' disease is, as most authorities supposed, a nervous disturbance—a functional derangement of some nerve centre or centres—possibly of some centre or centres in the floor of the fourth ventricle, or in the neighbourhood of the restiform bodies.

The result of excision of portions of the thyroid gland in cases of exophthalmic goitre seems to lend some support to the theory which has just been advanced; for in two cases in which the operation has recently been performed by Dr Lemke of Hamburg, remarkable improvement is said to have occurred.[1] Further, it is interesting to note that in both of these cases the exophthalmos disappeared and the heart's action became quiet and regular. If Lemke's results are corroborated by future observers, the theory which has been advanced above will have to be extended, and we

[1] *Deutsche medicinische Wochenschrift*, Jan. 8, 1891, p. 47.

shall, I think, be obliged to conclude that the exophthalmos and the increased frequency of the heart's action, as well as the other symptoms, result from the increased or perverted function of the thyroid. But even if this be granted, it may still, I think, with some probability be inferred that the primary cause of the disease (*i.e.*, of the hypertrophy or perverted functional activity of the thyroid gland) is due to a derangement of some part of the nervous apparatus.

The President invited discussion on the contrast which Dr Bramwell had drawn between myxœdema and exophthalmic goitre. The paper was an extremely suggestive one, and the study of differences between two diseases that were attended with affections of the same organ was very likely to lead to more correct ideas in regard to each. He (the President) was quite prepared to believe that myxœdema originated in some condition of the nervous system rather than of the thyroid gland, because in cases that had come under his observation there was atrophy of the ovaries and uterus. He had referred to this in a paper on Super-involution of the Uterus, which explained the amenorrhœa which Dr Bramwell had correctly described as likely to be present in myxœdematous women. As to the menorrhagia in exophthalmic goitre, he had seen it in some cases, but was not prepared to regard it as such a constant symptom in that disease as was the amenorrhœa in myxœdema.

Professor Greenfield congratulated Dr Bramwell on having found so interesting a subject for discussion. He had been in the habit for some years past, both in clinical and pathological teaching, of pointing out the antithesis between myxœdema and exophthalmic goitre, an antithesis which extended not only to the clinical facts, but also to those of morbid anatomy so far as he had been able to observe them. In exophthalmic goitre the condition usually present in the thyroid was comparable to an acute catarrhal process, with great increase and altered character of the epithelial cells, apparent proliferation throughout the gland, and great diminution of the formed colloid material. In other words, the essential change was one in which the process of secretion was greatly exaggerated and altered in character, whilst in myxœdema it was almost entirely abolished owing to atrophy of the glandular structures. In other respects, too, the morbid anatomy afforded a striking contrast. And seeing the inconstancy of the other pathological changes in exophthalmic goitre, he was led to believe that this must probably be the essential factor in the disease, especially in view of the converse set of facts afforded by myxœdema. In what manner the other phenomena were produced was yet open to investigation. In this view it seemed that more attention should be paid in treatment to direct action on the nutrition of the thyroid; and failing this by means of drugs, electrolysis or partial excision

would seem justifiable. The view that the disease must be one primarily of the nervous system had tended to withdraw attention from the importance of the changes in the thyroid gland. Of course, both in myxœdema and exophthalmic goitre, the primary cause of the changes in the thyroid was as yet unknown, and it might prove to be nervous, but upon this point there was no evidence of value. As to the symptomatology, amenorrhœa was in his experience far more common in Graves' disease than menorrhagia.

Mr Duncan said that from the surgical aspect experience would lead to the belief that in both diseases the symptoms were due to alterations in the thyroid. In myxœdema the results of experiments on the lower animals and extirpation in man pointed in this direction, although there were difficulties in the fact that apparently total ablation had been performed, as in a case of his own, without the sequel of myxœdema. He had used electrolysis in three cases of exophthalmic goitre. One was cured, one much improved in general symptoms, and the third was his only death in connexion with the operation of electrolysis. It was the result of fright, pure and simple. Before the operation the pulse was nearly 200 in the minute. He expected that the anæsthesia would relieve, and it did while she was under it. But on recovery the pulse became simply uncountable, and ran itself to exhaustion in two days. While the vascularity of these tumours might not in the interior be more than large, the arteries were greatly dilated and the surrounding venous plexus was most formidable. Still there was nothing in the hæmorrhage to prevent a well-planned operation from being easily enough performed.

Dr Affleck thought that although the discussion had wandered somewhat from the subject of the paper, no one would regret this after listening to the interesting statements which had been made on many points connected with these two remarkable diseases. A contrast of these diseases was a not unnatural comparison to make when one remembered that the element of the thyroid gland had to be considered in the pathology and clinical history of both. Even to the superficial observer the contrast as regards the *facies* of each was most striking; but when the chief points in the symptomatology were set forth, as had been so well done by Dr Bramwell, the contrast was still more interesting and instructive. As regards myxœdema, Dr Affleck referred to the experience of Kocher and many others who had operated on the thyroid gland, as well as to the experiments of Horsley, all of which seemed to prove the intimate connexion between the loss or atrophy of this organ and some of the leading symptoms of the disease, *e.g.*, lowness of temperature, with a sense of coldness which he had never seen absent in the myxœdematous. On the other hand, whether any condition of this gland played any part in the production of exophthalmic goitre, as had been supposed, was very doubtful, in

view of the facts that this latter disease may exist without appreciable affection of the thyroid, and that its symptoms are occasionally developed quite suddenly, as has been noticed by Trousseau and others, thus strongly suggesting a neurotic foundation. Vaso-motor abnormalities were prominent features here, and the occasional rises of temperature and perspirations bore testimony to this, no less than the vascular disturbance so constantly present. Dr Affleck had seen at least three cases of Graves' disease prove fatal by intractable diarrhœa, most probably of vaso-motor origin.

Mr Caird referred to the presence of myxœdema following the removal of goitre in the hands of Kocher, and the absence of this phenomena in the clinic of Socin. He also desired to know if myxœdema had followed extirpation of exophthalmic goitre.

Dr Bramwell stated, in reply, that his paper did not attempt to cover the whole subject of the relationship of the thyroid to myxœdema and allied diseases, nor to discuss the treatment of any of these conditions, it simply was intended to draw attention to the marked contrast which existed as regards the symptoms between myxœdema and exophthalmic goitre. He was not aware that Professor Greenfield had been in the habit of directing attention to these differences, nor did he know of any published observations in which the contrast was drawn in the way in which he had attempted to draw it. Dr Bramwell then referred in some detail to the results of excision of the thyroid both in man and the lower animals. He thought the evidence was conclusive that in man, as in the lower animals, the *complete* removal of *all* thyroid tissue resulted in many cases in the production of myxœdema; possibly careful protection from cold might prevent the development of such symptoms, just as it did in some of the lower animals.

Meeting VII.—April 8, 1891.

Dr A. G. Miller, *Vice-President, in the Chair.*

I. Exhibition of Patients.

1. *Dr Alexander James* exhibited a case of TETANY, on which he made the following remarks:—

Isabella D., æt. 21, residing at Leith, was seen and examined at the Infirmary, March 22nd, 1891, whither she had come to be treated for what she called "cramps" of both hands.

She is a well-nourished, healthy-looking girl, living with her parents in comfortable circumstances, and her work was ordinary house-work. With the exception of attacks of neuralgia during the winter of 1889-90 she had had no previous illnesses, and as a cause for her present state no depressing agencies, physical or

mental, can be elicited. It is noteworthy, however, that she has a somewhat enlarged thyroid gland. For some weeks before last New Year and since, she states that she has been feeling a numbness in her hands and arms, especially the right. This numbness has been felt every day, more or less, since the New Year, and seems to come on or get aggravated whenever she goes out into the air. She also feels numbness in the face, especially in the eyes and upper lip. The lips then feel as if stretched over the gums.

In addition to this numbness she experiences, when exposed to cold or when excited, the so-called "cramps," *i.e.*, the condition of tetany in her hands and arms, and to a less extent in her feet and legs. These cramps have been coming on nearly every day since the New Year, and lasting for two or three hours, and she states distinctly that the numbness does not always precede the "cramps," and that the "cramps" sometimes occur without numbness. They occur mostly in the morning, but sometimes at night, and their onset during sleep has sometimes awakened her.

When the cramps are present the hands show the characteristic position of tetany. The fingers are straight,—flexed, however, somewhat at the metacarpo-phalangeal joints,—and all slightly inclined to the ulnar side. The thumb, also straight, is drawn strongly into the palm, the hand assuming, as aptly described by Trousseau, a shape like that of the hand of the accoucheur when he introduces it into the uterus. The muscles of the forearm feel hard and contracted, but the patient, during the existence of the cramp, can still move the fingers to some extent. There is also, when the tetany

is present, great vasomotor dilation, so that the hands are darkly congested, as in the asphyxia stage of Raynaud's disease, and the patient states that during the "cramps" they sweat a good deal.

In the feet the toes are bent towards the sole. The dorsum of the foot is strongly arched and the heel pulled up by the muscles of the back of the leg. There is also some tendency to vasomotor dilation, but the symptoms as a whole are much less pronounced than in the arms.

These "cramps" seem to come on readily when the parts are exposed to cold, and pressure on the nerves and vessels of the limb seem to some extent also to bring them on.

There is no anæsthesia, and on testing the electric reactions of the affected muscles it was found that with the interrupted current their irritability was slightly less than that of the muscles of a healthy individual examined at the same time (*i.e.*, contraction occurred in her case when the secondary coil was at $5\frac{1}{2}$ cm. from the primary, as compared with 7 cm. in the case of the healthy individual). With the galvanic current the order of contraction was the normal one, viz.,—Kcl, Acl, Ao, Ko; and with 15 cells of the battery Kcl. produced slight tetanus.[1]

She is being treated with rest, fresh air, quinine and iron, and at present (April 1st) the disease appears to be running a fairly satisfactory course.

2. *Dr Shand* exhibited a specimen of STUMP HEALED BY THE FIRST INTENTION.

II. Exhibition of Specimen.

Dr Wm. Russell showed a fresh specimen of ANNULAR STRICTURE OF THE COLON AT THE SPLENIC FLEXURE which completely occluded the gut; the stricture was evidently malignant, but quite circumscribed, and there were no secondary deposits. This was a common form of intestinal stricture, and if it could be diagnosed during life it seemed to him that such cases were specially favourable for removal of the affected part.

III. Original Communications.

1. RESTORATIVE TREATMENT OF VARICOSE VEINS.

By William Taylor, M.D., F.R.C.P. Ed.

Eight years ago I had my second—and, I trust, final—attack of gout. I then realized to the fullest extent the accuracy of the description of that complaint given by an Edinburgh gentleman

[1] The accompanying woodcut is taken from a photograph, and at the time the photograph was taken the hands were not so markedly affected as usual, and the feet were hardly affected at all. It shows fairly well the enlargement of the thyroid gland.

to his medical adviser. "At eleven last night I went to bed as hale and hearty as you are, Doctor, and at two o'clock this morning I awoke with my foot in hell." Unfortunately my case presented an unusual aggravation, for both feet were affected. My left foot, although swollen and strained, was unable to accommodate the whole of the torturing elements, and the locked-out residue held an overflow meeting in my right, where, owing to the crush, the usual boundaries were again overstepped, and the heels encroached upon in addition to the toes.

Fortunately my head was clear, and my sympathies perhaps brightened, for I thought of the many poor men the pain of whose single feet I had so often tried to alleviate; and as the phantom procession glided past, I endeavoured to remember the applications which seemed to give most relief to the different individuals, and I determined to try them in turn on my own poor feet—a different one on each, so that I might the better judge of their comparative efficacy. On the third day my right foot was in heaven, but the left remained in the old place. However, I had discovered the remedy, and speedily applied it to the left foot, but, alas! it was vain. I had expected too much; and, forgetful of metastasis, I had attributed the cure to the wrong cause. The relief given to the right foot was simply the result of an adjournment of the fiendish meeting. My left foot remained obdurately insensible to the slightest alleviation from any or all of the anodynes employed. I shall therefore not enumerate them, for they were utterly useless. The complaint dragged its slow length along, and convalescence at last set in. My left foot regained its normal form and colour and freedom from pain. One of the remedies applied to this foot was a fly blister, which was meant to relieve the pain in the heel, but did not. Some weeks afterwards the site of the blister continued to be marked by a white patch, which, appearing in the midst of a mottled area of minute varicose veins, was somewhat conspicuous. I examined it carefully, but could not find any distended vein within it. Why not blister the remainder of the mottled area? I said to myself; and being very much at leisure, and utterly sleepless at any rate, I at once proceeded to do so. The blister rose, the mottling disappeared. I then continued the blistering process in the course of the internal saphena, which had for twenty years and more been in a prominent and painful varicose condition. This vein also disappeared. The raised blue distorted outline was no longer visible.

I had, like others, frequently had occasion to notice a great improvement in the condition of a varicose vein after an attack of phlebitis, and bearing that in mind, I felt that my present procedure was something like producing an artificial phlebitis, both in its appearance and results. I also thought of the contraction which shrivels up cutaneous structures after burns, and I felt that I was making use of a modified kind of burning which was followed by

similar contractions beneath the skin, and that these formed a kind of subcutaneous vitalized bandage which served to keep the vein within its proper limits. No doubt this is in some measure what did happen, but how? What are the changes involved in the process? In order to find assistance in determining this, I exhibited my leg to an eminent anatomist and physiologist in Edinburgh, and to an equally eminent surgeon and pathologist in London, but neither of them would venture to give an explanation of the anatomical condition then existing. The surgeon's remark was characteristic, and is almost enough to reveal his identity: "I don't know what has taken place in that leg, because I have not yet dissected that vein." "The treatment and its results are to me entirely new."

A correct theory seemed therefore hopeless without a foundation of anatomical fact on which to build it, and as no opportunity of dissection presented itself, I had to wait for more light, and content myself with the reflection, that whereas I once suffered from varicose veins, these sufferings are now relieved.

Meantime, being satisfied as to the absolute safety of the treatment, I lost no opportunity of giving my patients the benefit of it, and the results have been in every way satisfactory. This was peculiarly noticeable in aged persons, say of 60 and upwards, for they were of course the least promising class. In them the veins are more distended, thinner, and more relaxed than in the young, and the vitality of both the vessels and the neighbouring textures correspondingly diminished. Yet in every case the treatment has been successful, so that it is well entitled to be considered as eminently *restorative*.

Before such a Society as this I deem it unnecessary to enter into elaborate historical details regarding the various modes of treatment adopted for the cure of varicose veins. These are all familiar to you under the designations of Radical and Palliative. So far as I can see, the plan of blistering partakes of the merits of both without the danger of either.

The *Radical* treatment, which consists in the operation for obliteration, carries in its train such an array of misfortune as to entitle it to the designation hazardous. It is often only partially successful in obliterating the vein, but when completely so, it only results in a mutilation, for it removes an organ whose increased usefulness had in the first instance led to hypertrophy and distension of its walls. Its natural functions had been strained by overfeeding from the increased supplies yielded by the surface of the overworked muscles of the calf. Hence the greater frequency of varicosity in those who have to stand and lean forward over a counter or a wash-tub. The removal of the vein, therefore, without removal of the conditions which give rise to the disease is unscientific, for the delegation of the superficial vein throws increased work upon the deep-seated intermuscular veins whose functions are already pro-

portionately strained, and which are themselves often simultaneously involved in the primary diseased action.

The *Palliative* treatment is invariably represented by that most deceiving of all deceivers, the elastic stocking. In theory it is good; in practice it is bad. I speak from a prolonged personal experience, but this is not necessary to make its defects apparent. I had abandoned it long before I was cured, and had substituted a well-knitted, well-fitting woollen stocking, which was always clean, always comfortable, afforded a sufficient and consistent support, and did not deceive me into a belief in security which was fallacious. I never encounter an elastic stocking without ordering its immediate removal. In hot days they are too tight, and irritate the skin; in cold days they are too slack, and pucker into unequal folds. If you walk much, they gravitate towards the ankle and become irregularly hardened with perspiration. If you walk little, the ankle swells and the stocking becomes an obstruction to the circulation. This all applies to new, well-fitting stockings. What shall we say of old ones?

I have made use of the word *restorative* in preference to the term palliative; and although I have assumed that what takes place is the formation of crossing bands of connective tissue over the vein, constituting a subcutaneous binder, I do not by any means think that this is all—for if so, the analogy with phlebitis would not be complete. There is good reason to suppose that the coats of the veins themselves participate in the restored vitality, for their restored functions continue unimpaired for years. They are in no case obliterated; and this is best proved by the fact that after a limited number of years they again become distended and require a renewal of the treatment. This is not to be wondered at; for we cannot pretend that the restored vein is made better than it was originally, and if the sound vein gave way in the first instance, it is only reasonable to expect that under similar conditions the restored vein will also yield. This restorative treatment has the additional advantage of being applicable in regions where the ordinary palliative measures cannot be used, as in the walls of the abdomen and elsewhere. It is also suitable for dispelling the effusion and solid œdema which so often accompany the varicose condition.

The timely application of a series of blisters disperses this, and averts the almost certain sequel of hideous, intractable ulcers. The cure of these ulcers is most successfully accomplished—as pointed out by Mr Syme—by the application of blisters to the surrounding œdema. I always feel that in anticipating the formation of these ulcers and preventing it by dispelling the nidus of solid œdema where they form, I am acting consistently with the teaching of that great master.

Details of Treatment.—I first ascertain, and, if possible, remove the cause. I then prescribe such remedial measures as obviate a

tendency to renewal of the cause. Third, I place the patient recumbent, with the affected part elevated on pillows, for twenty-four hours, or such period as may be necessary to enable the limb to regain, as nearly as possible, its normal calibre. I then blister from the foot or sound part of the vein upwards along its course, say six inches daily, always watching for the first symptom of disturbance in kidney or bladder. This does not occur so readily as one would expect from the proximity of the blistering agent to the surface of the veins. This may probably arise from these having in a great measure lost their absorbing power, or from the suction necessary to absorption being absent. Still the bladder does sometimes become affected, and when this occurs we must suspend operations for a day or two. Certain of the blistering liquids contain camphor, and this is said to prevent strangury.

In order to make sure of this safeguard it is well to begin by a coating of Rubini's tincture, allowing it a minute or two to dry, and then applying the blistering liquid over it, with which it amalgamates. When the blistering liquid has had a minute or two to dry, I apply a coating of flexile collodion. This serves the double purpose of preventing evaporation and contracting the skin over the vein, and does not prevent, but rather aids the action of the blister. The blister *must* rise and the serum be withdrawn, but the action of the blister continues for days after, so long as any redness remains. The rising of the blister is useful in a secondary way, because the pain connected with it tends to reconcile the patient to the recumbent posture, which is absolutely necessary to success. When the whole of the affected vein has undergone blistering, the part should be carefully strapped with strips of adhesive plaster, 2 or 3 inches wide. This gently stimulates the whole surface of the leg, and assists the veins to resume their suspended functions. When the plaster begins to pucker, it should be renewed and fresh strips applied. Two weeks after the strapping has been applied it may be removed and the veins tested by placing the patient in the erect position. If the vein bulges anywhere, that part should be again blistered and the process renewed. If not, fresh strapping should be applied, and the patient allowed to take gentle exercise for a couple of weeks, when the leg should be again examined and the strapping renewed. Sometimes if the avocations of the patient compel him to walk earlier than is indicated, a solid coating of collodion over the affected surface may advantageously take the place of strapping. Thereafter, with renewed precautions as to avoiding any probable cause of obstruction, we may take leave of our patient with the parting advice, that he should daily practice walking on tiptoe until he acquires the habit of putting the toes down first. This saves the vein from the distending effects caused by the checked momentum of the contained column of blood which results from the jerk of placing the heel down first. For the same reason he should avoid

stepping on to or off a car when in motion, and leaping down from any height. He should also take care, on all suitable occasions, to elevate the legs during intervals of repose.

The Chairman said that the Society was much indebted to Dr Taylor for his interesting and practical paper. He had tried the method on a well-marked case of varicose veins with phlebitis in his wards in the Edinburgh Royal Infirmary with marked benefit. He thought that the blister acted by stimulating the part and promoting absorption.

Dr Allan Jamieson remarked that in connexion with so important and so common an ailment as varicose veins, any method of treatment which promised even cure for a limited time would be hailed with satisfaction. The causes of varicose veins were two-fold—mechanical and constitutional. Some persons suffered from them who did not habitually maintain the upright position. They were met with in children and young persons, and on parts of the body, such as the arms, not kept pendent. He thought that blisters acted beneficially by stimulating the peripheral circulation, and thus increasing metabolism. Dr Taylor had done wisely in thoroughly testing his plan before making it public; he had done well not to spring it on the notice of the profession without very careful trial; it might be well if this example were more generally followed.

Dr Shand said that he quite agreed as to the value of the paper read, especially as being the author's experience in his own person; but at the same time he felt in honesty bound to protest against the condemnatory views he had expressed regarding the elastic stocking treatment in varicose veins, especially as he had at the present time two cases of most satisfactory success of the mode of treatment. No doubt care and patience were sometimes taxed in the fitting and adaptation of suitable stockings, and even sometimes a preparatory bandaging, and where the skin is sensitive the application of some medicament with tannin for a little. Only the other day his older patient, a firm, stout, old housekeeper of a certain age (60) walked smartly two miles to and from church without difficulty, although two years ago she had had hæmorrhage from one vein, but was now all right with the help she obtained from the elastic stockings. He felt, when the subject was under discussion, he had no title to withhold such a defence as he now made for the utility of the elastics. In conclusion, he believed Dr Taylor's method would be an extremely useful resource in many unyielding cases, and he only hoped other Fellows would follow his example, and contribute their quota of practical experience, and especially personal experience, in professional treatment of diseases.

Dr W. Russell asked if any of the younger surgeons present could offer any explanation of the mode in which the blistering

acted? It was to be assumed that the facts were as stated by Dr Taylor; while his explanation that the amelioration was the result of the formation of fibrous bands which supported the distended veins, and that these bands were developed by the influence of the blister, could not be accepted.

Dr James asked what, in the opinion of any of the surgeons, was the effect of an attack of erysipelas on a leg affected with varicose veins? This might be of interest in connexion with the explanation of the beneficial effects of blistering in such cases.

Dr Allan Jamieson (in reply to Dr James's question) said that the effects of erysipelas were exerted on the lymphatic system in particular. Erysipelas consisted mainly in an inflammation of the lymphatic spaces, and the consequent production of a solid œdema. This was well seen on the leg in elephantiasis, and on the face, as had been shown by Mr Hutchinson, where the lips got thick and the tissue under the eyes was swollen in consequence of repeated attacks of erysipelas. He had not specially studied its influence on varicose veins.

Dr George Hunter concurred with the previous speakers as to the practical value of Dr Taylor's paper, and thought an explanation of the *modus operandi* of the remedy might be found in the following considerations:—As the result of the counter-irritant, inflammatory action will be set up in the skin (which is often very thin) immediately over the vein affected. This is communicated through the thin layer of subcutaneous tissue to the coats of the vessel. In these proliferation of the connective tissue corpuscles takes place, and contractile fibrosis of the unstriated muscular coat will naturally follow. It is evident that unless the calibre of the vessel be considerably reduced by contraction of the tissues surrounding it, the varix would be as great after the vesication as before it.

Mr Caird drew attention to the beneficial results obtained by the extirpation of two or three inches of the internal saphena vein as carried out by Lister several years ago, and more recently by Trendelenburg. He indicated the manner in which the importance of the rôle played by backward pressure might be demonstrated.

Dr Taylor thanked the Chairman and the Meeting for the very kind, he might almost say indulgent, reception they had given to his paper, and for the light they had thrown by suggestion on the probable changes which take place. Unfortunately no satisfactory explanation of the restorative processes which take place in and around the vein can be obtained without post-mortem dissection, and the object of the treatment is to delay that event as long as possible, so that we have to fall back upon our reasoning powers for an explanation. The theory started by Dr Hunter, he hoped, was the right one, as it seemed to be reasonable. Dr Allan Jamieson drew attention to what might be called the extrinsic and intrinsic causes of varix. Dr Taylor thought that, apart from mechanical

causes, the constitution most obnoxious to it was the gouty. There seems a greater tendency to varix in that constitution than all others put together. You find it taking place in the form of precordial vascularity, and at other sites where there seems to be no obstruction or mechanical means to account for it. The remarks of Dr Caird were extremely interesting as showing how to select cases suitable for operation; and if these were limited to such as he described, there would be greater success attending it. He hoped the challenge thrown out by Dr Russell would yet bear fruit. It was extremely difficult to get men to state a theory, and he was therefore the more grateful to those gentlemen who had to-night contributed so freely in that direction.

The Chairman, in answer to Dr James, stated that the only result on varicose ulcers of erysipelas that he had noted was that they were made much worse and more difficult to heal. As to the effect on the varicose veins themselves he could not speak, not having directed his attention to that point.

2. THE RELATIONS OF THE ABDOMINAL VISCERA IN THE INFANT.

By J. W. Ballantyne, M.D., F.R.C.P. Ed., Lecturer on Midwifery and Gynæcology, Medical College for Women, Edinburgh; and on Diseases of Children, Minto House, Edinburgh.

The conclusions with regard to the anatomical relations of the abdominal viscera in the new-born infant, which are stated in this communication, are founded upon the examination of seven full-time infants and one seven months' fœtus by the frozen sectional method, and also upon the simple dissection of several other infants. The method of investigation by means of frozen sections is now so well known that it is not necessary to describe it here; but it may be said that in all the cases, save one, the infant was placed in the freezing mixture in the dorsal position. Drawings were obtained from the sections by means of gelatine plates.

The relations of organs and structures in the abdomen of the infant differ in several particulars from those found in the same region in the adult, and it is therefore very necessary that the physician, especially the pediatric physician, should have in his mind a very clear conception of the anatomy of the abdominal viscera in the infant; and, further, this region must be of interest to him for the reason that children are particularly liable to diseases affecting the intestines, the stomach, and the mesenteric glands. A knowledge of the peculiarities of the abdominal viscera is also essential to the surgeon who may be called upon to operate for imperforate anus, for hernia, or for other abnormal conditions in the newly-born infant.

The comparatively small size of the face, thorax, and pelvis of

the infant gives to the region of the abdomen the appearance of disproportionate largeness, but the abdomen is also really voluminous on account of the large size of the liver. It is not uncommon to find the abdomen pathologically enlarged in children from tabes mesenterica, ascites, distension of the bladder, etc., and among the specimens dissected by me there were examples of distension from ascitic fluid, from peritonitic effusion, from partly coagulated blood due to rupture of the spleen, and from splenic hypertrophy. One of the specimens of which frozen sections were made showed also an over-distended bladder, which caused an undue prominence of the lower part of the abdomen.

The palpation of the infant's abdomen reveals the presence of a hard, firm body projecting downwards from under the cover of the ribs to the level of the umbilicus. This body is the liver, and its large size must be borne in mind in palpating and percussing the infantile abdomen. The bladder, also, which is practically entirely an abdominal organ at birth, will, if distended, give a dull area in the hypogastric and lower umbilical regions.

At birth, and for fully a week afterwards, there is found attached to the abdomen the portion of umbilical cord left after ligature and section. The point of insertion of the umbilical cord into the anterior abdominal wall of the infant differs in its position from that found in the adult condition. It is usually stated that during the first two years of life the umbilicus occupies the central point of the body (M'Clellan, *Keating's Cyclopedia of Diseases of Children*, i. p. 30); but I have found it more commonly to be placed a little nearer to the feet than to the cephalic vertex in the infant. If the total length of the infant be 50 ctms., then the umbilicus will be distant from the vertex 27 or 28 ctms. As the child grows older, and as the legs lengthen, the central point of the body is found to coincide with the pubes. In its relation to the vertebral column, also, the umbilicus differs somewhat in the infant when compared with the adult. It is, in the normal infant, found to coincide in level with the disc between the fourth and fifth lumbar vertebræ, and to lie at or a little above the level of the highest point in the iliac crests. In the adult the umbilicus is situated opposite to the lower border of the third or the upper border of the fourth lumbar vertebra, and is, therefore, fully one vertebra higher than in the infant. In an infant with dropsy the umbilicus lay at the level of the first sacral vertebra; but in this case its low position was evidently due to the distended, slightly pendulous condition of the abdomen.

On the internal aspect of the anterior abdominal wall in the infant the component parts of the umbilical cord can be distinctly seen. The umbilical vein passes upwards in the falciform ligament of the liver, and sometimes shows a rounded swelling (containing blood-clot), about the size of a marble, in its course: the two arteries of the cord pass downwards to the bladder and diverge as the hypo-

gastric arteries to join the internal iliacs, which in the infant appear as if they were branches of the hypogastrics; and the urachus passes downwards in the middle line to the apex of the bladder.

Certain outstanding peculiarities in the relation of the abdominal viscera are revealed when the abdomen is opened. The very delicate transparent character of the great omentum is noteworthy, but the most striking feature is found in the large size of the liver (Plate IV.) Roughly speaking, it may be said that the liver occupies nearly one-half of the abdominal cavity. Ribemont demonstrates its position and size as follows:—A plane passing along the inferior surface of the liver divides the abdomen into two compartments, pyramidal in form and nearly symmetrical. The one has its base above, occupying the right hypochondrium and the epigastric region, whilst its apex is turned towards the iliac crest of the same side; this compartment contains the liver. The other, with base inferior, and apex turned towards the posterior part of the left hypochondrium, contains the intestinal coils, the spleen, and the stomach. The liver extends vertically from the diaphragm to a point some millimetres above the right iliac crest, and from the right hypochondrium to the left in a transverse direction. Ribemont's description in the main agrees with what the dissections and frozen sections show; but I have not in any case found the lower limit of the liver to be so near to the right iliac crest as he makes it. Doubtless there are considerable individual differences in the size of the liver within the limits of health in children as in adults.

Upon the subject of the *form* of the liver the sectional method by freezing has thrown an immense amount of light. Dr Symington, in a masterly paper on the liver in the adult, read before this Society in the December of 1887, showed that most erroneous notions prevailed with regard to the form and surfaces of the liver. When removed from the body and laid upon a plate, the organ seems to have only two surfaces, an upper and an under; but when hardened by freezing and studied *in situ*, the liver at once appears with at least five surfaces. The liver may be roughly compared to a jelly made by a cook which has not set properly, and which when turned out upon a plate collapses: no one can tell the original form of the jelly from the flattened mass on the plate. As seen in frozen sections, sagittal and coronal, the liver is found to possess five surfaces—a posterior, pointed out by His in 1878; a superior; an inferior; and an anterior and a right, as demonstrated by Symington in his paper above referred to. The inferior surface is more correctly termed left inferior, for it is directed not only downwards but very markedly towards the left side. Now, these hepatic surfaces which are present in the case of the adult liver are all particularly well defined in the case of the infantile organ, and can be recognised with the greatest ease. Some of my sections have led me to describe a left surface in

addition to the others, but I am not certain that this surface is constantly present in all infants.

Looking upon the liver, therefore, as a "right-angled triangular prism with the right angles rounded off," we find that it shows five, or in some cases six, surfaces. From the consideration of sagittal, coronal, and transverse sections of the abdomen, and from the subsequent building up the liver from the separate sections, the relations of the various surfaces can be accurately described. The *superior surface* (Plate I.) is in contact with the diaphragm, to which it is accurately moulded, and has therefore a general convexity with a local concavity immediately underlying the heart. One of my sections might appear at first sight to be opposed to this statement, for in that specimen the left portion of the superior hepatic surface is as high on the left as on the right side; but in that case there was a hypertrophied spleen, which had pushed up the liver on the left side. The superior surface is in infants very clearly marked off from the anterior surface, from the small posterior, and also from the left inferior surface; but the line of demarcation dividing it from the right surface is not so easily determined (Plate III.)

The *anterior hepatic surface* (Plates I. and II.) is a clearly marked one in the infant, and is divided from the left inferior surface by the thin so-called anterior border of the liver. This surface has, in the case of the adult liver, had its form compared to that of a right-angled triangle; but in the case of infants in whom there is a left hepatic surface the anterior surface of the liver is more quadrangular than triangular in shape. Even if the left hepatic surface be merged in the superior surface, it will still mask the triangular form of the anterior area in the infant; in the adult the left surface, on account of the relatively small size of the left lobe, is non-existent, and therefore the anterior aspect of the liver has a triangular form, the rounded right angle of which marks the meeting-point of the right and superior surfaces, whilst the hypothenuse of the triangle passes downwards and towards the right side. The anterior liver surface in the normal infant corresponds in its vertical extent in the middle line with the ninth, tenth, eleventh, and twelfth dorsal, and with the first and second lumbar vertebræ, and its free border reaches to within 2 ctms. of the umbilicus. On the left side of the middle line the vertical extent of the anterior surface gradually diminishes in area, whilst on the right side it gradually increases, so that the border of the liver there comes to within 1 or 1·5 ctm. of the right iliac crest.

The *right surface* (Plate III.) of the liver is the least clearly defined, for it is separated from the other surfaces (except the left inferior) by rounded, ill-defined borders, nevertheless it is present in both the adult and infantile organ, although it is of much larger extent in the case of the latter. The *posterior sur-*

face (Plate I.) is small, and is, like the right, not very clearly marked off from the others. It is, however, very evident in sagittal mesial sections. According to His, it includes the notch for the œsophageal end of the stomach, the posterior part of the longitudinal fissure containing the ductus venosus, the groove for the inferior vena cava, the lobus Spigelii, the depression for the supra-renal capsule, and that part of the right hepatic lobe above the kidney which lies in direct contact with the liver. It is a small surface, and corresponds in vertical extent with the tenth, eleventh, and twelfth dorsal vertebræ in the infant.

The *left inferior surface* (Plates I. and III.) is very clearly marked off from the others by the so-called anterior border of the liver, and by the groove anterior to the lobus Spigelii. It is a large surface, and is in relation with many organs which leave impressions upon it easily recognisable in frozen sections and in hardened specimens. The organs in contact with this surface are the stomach, the spleen, part of the supra-renal bodies, the duodenum, transverse colon, the hepatic flexure of the colon, and some of the coils of small intestine lying in the upper part of the abdominal cavity. On this surface lie, as every student of anatomy knows, the longitudinal and transverse fissures, and that for the gall-bladder; and the area is made up of the under surfaces of the right and left lobes, and of the quadrate and caudate lobes of the liver. The *left* hepatic surface (Plate III.), which is sometimes, but not always, found in the infantile organ, is ill-defined, and has, for the sake of comparison with the adult liver, been merged in the upper surface.

The relative size of the various liver surfaces is of some importance, and in estimating their limits I have followed the directions laid down by Symington. In two normal cases the superficial extents of the various surfaces were ascertained to be as follows:—

	Case I. Ctms.	Case II. Ctms.
Ant. surface, transverse extent, .	9·5	9·5
So-called ant. border, . . .	15·0	15·2
Sup. surface, transverse extent, .	8·7	8·8
„ „ ant. posterior extent,	2·7	2·0
Right surface, vertical extent, .	5·5	4·8
„ „ ant. post. „ .	3·8	3·5
Post. surface, vertical „ .	3·3	3·0
„ „ transverse „ .	5·7	5·3
Left inf. surface, transverse extent,	9·2	10·0
„ „ „ ant. posterior extent,	4·2	3·3

From the foregoing table it will be seen that the anterior and left inferior surfaces were the largest; the superior, right, and posterior surfaces showed much smaller areas. The surfaces come in order thus,—anterior, left inferior, superior, right, and posterior.

With regard to the above description of the characters and extents of the various surfaces of the liver, it may be said, *Cui bono?* The answer is not difficult to find. If the clinical investigation of the abdominal viscera in the infant is to reach the same state of perfection as that in the adult, then it is necessary that the relations and dimensions of all the parts to be palpated or percussed be accurately ascertained in normal cases.

In Dr Symington's paper above mentioned allusion was made to displacement of the liver due to distension of the stomach: in none of my specimens was the stomach distended, but in one case there was hypertrophy of the spleen (Plate III., Fig. 2), and in that instance there was a displacement of the liver similar to that described by Symington. The liver as a whole was rotated towards the right side on a vertical axis passing through the inferior vena cava, and the left lobe of the liver was also pushed slightly forwards and upwards. The displacement was easily ascertained by comparing the line of attachment of the falciform ligament to the liver with the middle line of the body.

The lobes of the infantile liver differ in relative size from those in the adult organ. Both the right and the left lobes are very large, and the lobus Spigelii is of extremely variable dimensions. The longitudinal fissure is very large in the infant, and contains the umbilical vein and the ductus venosus. In one specimen there was found a very large pons hepatis bridging over the anterior part of this fissure.

In all the still-born infants dissected by me, and in one infant who died on the sixth day of life, the gall-bladder was found to be distended with bile (Plate IV.)

The liver in the infant has a darker colour than in the adult, and its weight in relation to the general body weight is as 1 to 18.

The explanation of most of the peculiarities in the anatomical relations of the abdominal viscera in the infant is to be found in the large size of the liver at that period of life.

The Stomach.—It has been frequently stated that in the case of the new-born infant that has not breathed the walls of the stomach are in close contact, the gastric cavity being only potential; but in all the cases examined by the sectional method there was a real gastric cavity containing frothy watery mucus (Plates II. and IV.) In the case of the infant that died six days after birth (Plate III., Fig. 1), the stomach contained milk, mucus, and air-bubbles; this infant died suddenly from heart clotting. In one of the still-born infants there was a small quantity of meconium in the stomach, and in this case there had been great delay in the labour, and forceps had been used. It is, of course, difficult to say how far post-mortem changes may serve to account for these conditions of the stomach contents, and medico-legal proofs of "live-birth" founded upon the state of the contents of the stomach are apt to contain fallacies.

The stomach is small in size at birth, and contains only 1 or 1½

oz. of fluid without being over-distended. During the first two months of life there is a rapid increase in gastric capacity, but thereafter the increase is slow.

Symington does not believe that the stomach in the new-born infant differs in form from that in the adult; but my specimens have led me to believe that the fundus of the stomach is relatively small at birth, and that the lesser curvature forms a more acute angle than in the adult organ. The upper part of the lesser curvature runs vertically downwards and slightly forwards, and then turns sharply round to the right, forming an angle which is smaller than a right angle. The vertical position of the lesser curvature is now by many anatomists regarded as normal even in the adult stomach, and yet it is still described in many text-books as the *upper* curvature. It is certainly not the upper border in the infant's stomach, there the fundus is distinctly the highest part of the viscus.

As regards position, in all the cases examined the stomach lay entirely to the left of the middle line, the pylorus being situated immediately in front of the body of the first lumbar vertebra. It must be remembered that in none of these cases was the stomach greatly distended. In all the specimens, also, the stomach lay entirely under cover of the liver; hence, on opening the abdomen of the new-born infant, one must not expect to see the stomach until the liver be raised (Plate IV.) The anterior relations of the stomach were with the left inferior surface of the liver, whilst posteriorly the viscus was in contact from above downwards with the anterior surface of the spleen, the left supra-renal capsule, the upper end of the left kidney, and with the tail and body of the pancreas. Below the greater curvature of the stomach was the transverse colon. The lesser curvature runs at first parallel to the left side of the vertebral column, and then turns, to pass transversely to the right side in front of the spine. The relations of the stomach with the left kidney are not extensive in the infant, for the large adrenal of that side intervenes.

The well-known ease with which an infant vomits has found many explanations; but, to my mind, it is chiefly due to the fact that the stomach is surrounded on all sides by firm resistent organs. In front lies the liver, and behind are the spleen, supra-renal body, and kidney, and therefore any increase in abdominal pressure will tell with greater effect upon the stomach thus surrounded than if it were in contact with the more resilient anterior abdominal wall. It may be also, as is suggested by Gubaroff (*Arch. f. Anat.*, 1886), that the valvular action of the cardiac end of the stomach is weak in new-born infants.

The Intestines.—The *small* intestine, according to Treves, measures 9 ft. 5 in. in length at birth, and grows about 4 ft. in the first two months of life; but it is very probable that the length varies rably in different infants. The duodenum is continuous

with the pyloric end of the stomach in the middle line of the body, and opposite to the body of the first lumbar vertebra. The duodenum, in its three parts, has the same relations with the pancreas as in the adult, for it forms a curve with an internal concavity in which lies the head of that organ. The third part of the duodenum crosses over the vertebral column at the level of the second lumbar body, and so reaches the left side of the middle line, where it becomes continuous with the commencement of the jejunum. The jejunum and ileum are less fixed in position than the duodenum. In the new-born infant they contain, as a rule, a little meconium, and they call for no further special notice.

The Large Intestine.—In the infants which were examined by the frozen sectional method there were several peculiarities in the arrangement of the large intestine, and it will be necessary to study these cases in some detail. The *cæcum* and *ascending colon* first call for description. In two cases (C and D) the cæcum was found in the position which it occupies in the adult; that is to say, in the right lumbar and right iliac regions. In case C (Plate III., Fig. 1), the cæcum was found lying a few mms. above the crest of the right ilium, at which point it became continuous with the termination of the ileum by the small rounded aperture known as the ileo-cæcal valve. It was firmly fixed to the anterior surface of the iliac fossa, and to the posterior abdominal wall in the right lumbar region above the right iliac crest. Thence the ascending colon passed upwards and towards the middle line to the immediate neighbourhood of the gall bladder on the under surface of the liver, where there was a feebly-marked hepatic flexure. This infant had lived for five days, and consequently the intestine contained only a small quantity of meconium, and was not in a distended condition. The vermiform appendix was more conical in form than in the adult condition, the base of the cone was attached to the bowel, the appendix was 3·1 ctms. in length, was twisted upon itself, and was firmly attached to the outer surface of the cæcum by a thin piece of mesentery. In case D the cæcum was situated in the right lumbar region, 1 ctm. above the level of the right iliac crest, and lay, therefore, at a higher level than in the preceding case. The ileo-cæcal valve was 5 mms. in diameter, and the ileum opened into the cæcum at an acute angle. The cæcum was largely distended with meconium (the infant was still-born), and was firmly attached by a short mesentery to the posterior abdominal wall on the right lumbar region. There was a well-marked appendix vermiformis coming off in a conical manner from the cæcum, it measured 4 ctms. in length, and was, as in case C, firmly attached to the cæcum by a thin mesentery. The appendix was also folded upon itself. The ascending colon passed upwards along the right lateral abdominal wall to the under surface of the right lobe of the liver at the border separating the right surface

from the left inferior. At this point there was a well-marked hepatic flexure. In this case, therefore, the arrangement of the cæcum and ascending colon closely resembled that found in the adult, with the single exception that the cæcum lay at a level slightly superior to that it occupies normally in the adult. In case C, whilst the cæcum was practically in the position in which it is found in the adult, the course of the ascending colon upwards and slightly to the left to the neighbourhood of the gall bladder and head of the pancreas, showed that this portion of the bowel had a position which is normal at the stage of intra-uterine life before the complete revolution of the large intestine round the axis of the superior mesenteric artery has taken place. In yet a third case (Plates I. and II.) a state of affairs still more closely resembling the embryonic disposition of the cæcum and ascending colon was found, for in this infant (also still-born) the cæcum lay in the middle line of the body at the level of the umbilicus in front, and of the fifth lumbar and first sacral vertebræ behind, and rested upon the posterior surface of the bladder, and was in contact with the anterior surface of the spinal column. The ileo-cæcal valve lay at the level of the lower border of the last lumbar vertebra. From the cæcum the ascending colon passed almost vertically upwards to the liver, with which it came in contact in front of the pancreas and first part of the duodenum, and there formed the hepatic flexure. In this case, therefore, the ascending colon lay only a few mms. to the right of the middle line, whilst the cæcum lay in the middle line, and extended slightly to the left side of it. The ileo-cæcal valve lay, as nearly as might be, in the exact middle line of the body behind the umbilicus. There was therefore here an arrangement of parts very closely simulating that found in the embryo before the cæcum and ascending colon of the primitive intestine have taken up their mature position in the right lumbar and right hypochondriac regions. It is usually stated that in the process of development the cæcum, after crossing over the small intestine, passes to the right hypochondrium, and then pushes downwards into its ultimate position in the right lumbar and right iliac regions; but my cases above recorded go to support the view of the development of the colon stated by Bruce Young (Cleland's *Memoirs of Anatomy*, vol. i. p. 81) in the following words:—"The portion of colon from which in normal circumstances the ascending and right half of the transverse colon are developed already exists as part of the primary loop, before the twist of the intestine to the right occurs; so that when the rotation does take place, it is the whole loop with its peritoneal surroundings which revolves round the superior mesenteric artery until the cæcum lies in the lower part of the right lumbar region. A slight downward growth is then all that is needed to carry it into the adult position." The above-named anatomist records the case of an adult male in whom the cæcum projected into the pelvis, and with the

lower part of the ascending colon lay free on the *left* side of the middle line. The ascending colon passed upwards on the left side of the middle line, and in contact with the anterior abdominal wall, to the upper end of the duodenum; it then turned abruptly to the left, descended as low as the left iliac fossa, and then passed upwards again in a series of closely-packed convolutions to reach the splenic flexure. The descending colon, he states, ran normally into the sigmoid flexure and rectum. In this case the loop of bowel descending from the neighbourhood of the duodenum to the left iliac crest was regarded as part of the ascending colon, and, therefore, according to this view, there was no transverse colon at all; but for reasons to be stated immediately, I am inclined to regard this loop as transverse colon displaced downwards and to the left. Dr Bruce Young's case taken in conjunction with the three cases just described represent gradations in abnormality from the degree in which the cæcum and ascending colon lie entirely to the left of the middle line to that in which there is only a slight displacement of the cæcum. In Bruce Young's case both cæcum and ascending colon lay to the left of the middle line; in my case A (Plate I.), the cæcum lay in the middle line behind the bladder, and the ascending colon passed upwards, lying only a few millimetres to the right of the middle line; in case C (Plate III.) the cæcum was approximately normal in position, but the ascending colon passed upwards and towards the left side till it reached the second part of the duodenum and head of the pancreas; and in case D the ascending colon may be said to have been normal in position, but the cæcum was situated at a slightly higher level than was normal in the adult. In the four cases there were, therefore, four stages in the process of rotation of the ascending colon and cæcum from their primitive position to the left of the middle line to their adult position in the right iliac, lumbar, and hypochondriac regions. Dwight also, in his *Atlas*, shows the cæcum in a three year old child, lying on the right side at the level of the umbilicus, and he says—"I doubt very much if, as a rule, the cæcum has reached its permanent position at birth, and think that not very rarely it does not reach it for a year or two afterwards." At the time when I first read the above statement I felt inclined to agree with Dwight in his conclusion, for I had then made frozen sections of five infants, in none of whom were the cæcum and ascending colon in the position which they normally occupy in the adult. Two of these infants, however, showed gross pathological conditions of the abdominal organs, and were, therefore, left out of account; and I had an opportunity soon afterwards of making sections of two other normal infants and one seven months' fœtus, in all of which the cæcum and ascending colon were quite normal in position. The only conclusion which it is, therefore, permissible to draw is, that the cæcum and ascending colon have frequently at the time of birth a position more or

less far removed from that found normally in the adult arrangement of parts. It is more difficult to judge from simple dissections of the abdominal viscera, for the parts are apt to be displaced artificially; but even in infants examined in this way I have not uncommonly found that the cæcum and ascending colon were more or less abnormal in position at birth.

The Transverse Colon.—In case D, that in which the ascending colon was practically normal in position, and in the other cases in which that part of the bowel showed no abnormality, the transverse colon also was normally situated. It began on the right side in the hepatic flexure, passed towards the middle line along the under surface of the liver, and was then directed to the right along the greater curvature of the stomach to reach the splenic flexure. It had a transverse meso-colon, and was also attached to the great omentum. In cases A and C (Plates I. and II.), in which the ascending colon was abnormally situated, the transverse colon found its commencement in a feebly marked hepatic flexure which lay not far to the right of the middle line in the neighbourhood of the gall bladder. It might have been expected, therefore, that in these cases the transverse colon would have a short course, and at first sight it was thought that this was so. The transverse colon formed a flexure in the neighbourhood of the border of the left lobe of the liver, then the bowel passed downwards to the left iliac crest, then it turned upwards again, reached the spleen, where it formed another flexure, and again proceeded downwards along the posterior abdominal wall, to become continuous with the sigmoid flexure. At first sight it seemed that in these two cases there were two splenic flexures, and two descending colons, a superficial and a deep; but the examination of the arrangement of the mesentery and of the relations of the bowel soon demonstrated the fact that the so-called superficial splenic flexure and descending colon were really a loop of transverse colon displaced downwards, whilst the deeper structures were in truth the splenic flexure and descending colon properly so called. The transverse colon, therefore, in these cases, did not pass simply from right to left hypochondrium, but formed also a loop passing down to the left iliac region, and lying superficial to the true splenic flexure and descending colon. In the infants in whom the ascending colon was normally situated, the transverse colon formed no accessory loop; and it is, therefore, reasonable to suppose that the abnormal arrangement of the transverse depended upon that of the ascending colon. The bowel was crowded into the left side of the abdominal cavity, the large left hepatic lobe barred its progress upward, and it therefore formed a loop passing downwards into the left iliac region, and displaced the small intestines to the right side of the abdomen. It cannot be said from these cases whether the above is a common arrangement or not; but it must at any rate be borne in mind by

abdominal surgeons that this disposition of parts does occasionally occur in the new-born infant.

The Descending Colon.—This part of the large intestine was normally situated in all my cases, and calls for little or no notice. It passed from the splenic flexure in front of and external to the left kidney down to the commencement of the sigmoid flexure, which was a somewhat variable point. It was attached by areolar tissue to the quadratus lumborum muscle, and to the left diaphragmatic crus.

The Sigmoid Flexure is large in the infant at birth. It becomes continuous at the level of the left iliac fossa with the lower end of the descending colon, and terminates, after having described one or two curves, in the first part of the rectum. In all the cases examined by the sectional method, a loop of the sigmoid flexure was found lying below the brim of the pelvic cavity and posterior to the bladder in the male and to the uterus and annexa in the female infant. The loop of the sigmoid flexure was usually situated a little to the right of the mesial line in the pelvis, and the fact that this part of the bowel was so constantly found in the narrow infantile pelvic cavity is most noteworthy. To its presence has been ascribed a very obstinate form of constipation in the infant, which has been named in consequence of this, "anatomical" constipation. I know not whether the presence of a loop of sigmoid flexure in the pelvis is a constant occurrence in infancy or not; but it is well known that this part of the bowel is relatively long at birth, and that it has a rather long meso-sigmoid.

The Rectum is that part of the bowel which is lodged in the pelvis, and it is not, therefore, an abdominal organ; but its chief peculiarities in the infant may be briefly stated. It is relatively larger in the infant than in the adult, it is more vertically situated in the pelvis, and it is more nearly a straight tube. In the infant, therefore, this part of the large intestine is more deserving of the term *rectum* applied to it than it is in the adult, where it is very far from being a straight tube. The large size, vertical position, and nearly straight character of the rectum in the infant make this part of the bowel very accessible for the thermometer, enema syringe, etc.

The Spleen.—Luschka describes the adult spleen as possessing three surfaces—a phrenic, a gastric, and a renal; but this description will not suffice for the infantile spleen. In the infant the liver comes into contact with the spleen behind and external to the stomach,—in fact, in one case (D) the spleen was entirely hidden by the left lobe of the liver, and lay in a concavity on the left inferior surface of that organ. Further, in the infant the spleen has a direct relationship with the left supra-renal capsule instead of with the left kidney. The spleen in the infant may, therefore, best be described as having four surfaces, and these may

be named gastric, hepatic, phrenic, and supra-renal. These surfaces are all clearly marked, and show the impressions of the organs which have lain in contact with them. Lateral sagittal sections serve to demonstrate these surfaces. Thus in a section made a little to the left of the middle line (Plate II.) three of the surfaces are seen—the phrenic posteriorly, the gastric in front, and the supra-renal inferiorly; but at a point further from the middle line all the four surfaces are visible—phrenic posteriorly, hepatic anteriorly, gastric antero-internally, supra-renal inferiorly. The antero-internal or gastric surface in relation with the tail of the pancreas as well as with the stomach. If it be deemed advisable, the surfaces may be named according to the direction in which they point. Thus the phrenic surface becomes the posterior, the hepatic becomes the antero-external, the gastric and pancreatic surfaces become the antero-internal, and the supra-renal becomes the inferior surface. Near the middle line of the body the spleen shows three surfaces, and therefore vertical sections here represent the organ as triangular. Further to the left, however, there are four surfaces, and the spleen is there quadrangular in vertical section. In one case (Plate III., Fig. 2) the spleen was much hypertrophied, and therefore in that case the antero-external surface came into contact not only with the liver, but also with the anterior abdominal wall, whilst the antero-internal surface was related to the stomach and pancreas, and also to the mesentery and splenic flexure of the colon, and to an accessory spleen (which was present).

The following table contains the measurements of the infantile spleen in two cases (A and D) in which it might be regarded as normal, and in one case (C) in which it was much enlarged:—

Surfaces.	Cases A & D. Ctms.	Case C. Ctms.
Antero-external or hepatic—		
Vertical extent, ...	4·0	6·1
Transverse do., ...	2·9	3·2
Length of anterior notched border, .	3·9	5·3
Antero-internal or gastric surface—		
Vertical extent, ...	2·8	5·5
Antero-posterior extent, . .	2·2	1·9
Posterior or phrenic surface—		
Vertical extent, ...	2·6	4·5
Transverse do., ...	1·4	2·0
Inferior or supra-renal surface—		
Vertical extent, ...	1·1	1·7

The antero-external or hepatic surface is, therefore, the largest, and it is interesting that in the infant at birth this large surface is under cover of the left lobe of the liver. As the stomach grows rapidly in size during the first weeks of life, the hepatic surface of the spleen is quickly encroached upon by the gastric surface of that

organ; but at birth the hepatic surface is the largest, the gastric comes next, then the phrenic or posterior, and the inferior or supra-renal is normally the smallest. In the case in which the spleen was hypertrophied, it is interesting to note that the large size of the organ was chiefly due to increase in a vertical direction.

The relations of the spleen with the surrounding viscera have been already in part described: the upper end of the organ reaches as high as the fundus of the stomach, *i.e.*, to the level of the 9th rib, near its attachment to the spinal column. The posterior surface rests upon the diaphragm, and behind the diaphragm is the base of the left lung. The relations of the spleen to the liver, stomach, transverse colon, and splenic flexure are well brought out in Plates II. and III.

The anterior border of the spleen is sharp and notched, and in most cases I counted four notches, two close together and two further apart. None of the notches were deep in the cases examined.

The spleen is darker in colour in the new-born than in the adult, and is very easily torn. The hypertrophied spleen was, however, much firmer than is normal at birth. The cause of the hypertrophy I am ignorant of. The infant had lived five days, and had died of ante-mortem heart-clot which may have had some connexion with the large size of the spleen.

The remaining abdominal viscera—the kidneys, supra-renal capsules, and pancreas—require only a few words of description, for they differ little in their characters from the same organs in the adult.

The *Supra-renal Capsules* are relatively large in the new-born infant, being usually equal in volume to one-third of the kidney. They have the form of a triangular pyramid; and they rest upon the upper end of the kidney, covering it like a cap. The base of the adrenal descends upon the kidney anteriorly to the level of the hilus of that organ, and is hollowed out in order to fit the renal convexity. The apex of the right adrenal lies between the liver and the right crus of the diaphragm, at about the level of the 10th rib; that of the left is wedged in between the spleen and the left diaphragmatic crus at a point a little above the level of the 11th rib. The posterior surface rests upon the diaphragm at the side of the vertebral column, and anteriorly the right adrenal comes into contact with the left inferior surface of the liver and with its posterior surface, whilst the left is related to the spleen, stomach, pancreas, and small intestine. Antero-posteriorly the supra-renal capsule measures, on an average, 1 ctm., and its greatest transverse diameter at its base is 1·5 ctm. Its vertical diameter from apex to anterior border of base measures 3·5 ctms., to posterior border of base 1·5 ctm. It therefore descends to a lower level in front of the kidney than behind it. The outer zone of the capsule is seen on section to be of a light pink colour, the inner zone is of a darker tint, and here and there fissures run into the organ, dividing it into two or three lobes.

The Kidneys.—These organs correspond in level with the 1st,

2nd, and 3rd lumbar vertebræ, with the disc between the 12th dorsal and 1st lumbar, and with that between the 3rd and 4th lumbar vertebræ. The right kidney measures about 3·8 ctms. vertically, the left about 3·5 ctms. The greatest antero-posterior diameter is from 1·5 to 1·8 ctms., and the greatest transverse about 2·3 ctms. It is usually stated that the left kidney is longer than the right, and is situated at a higher level in the abdomen; but I have not found this to be the case in the new-born infant. In position the kidneys were practically identical, and in vertical measurement the right was the longer. The relations of the kidneys to surrounding organs are practically the same as in the adult, with the exception that the supra-renal capsules cover a greater part of their surface, especially anteriorly. The hilus of the kidney lies at the level of the 2nd lumbar vertebra. The renal lobulation is more marked at birth than in later life, but is not so distinct as it is in the fœtus. The lower end of the kidney reaches to within a distance of a few millimetres from the iliac crest.

The *Pancreas* has the same relations with surrounding viscera and vessels in the infant as in the adult. In the middle line the head and commencement of the body of the pancreas lie opposite to the 1st and 2nd lumbar vertebræ. The left renal vein crosses immediately behind the pancreas, the superior mesenteric vein and artery lie partly behind it and partly in its substance, and the first part of the duodenum and the transverse colon lie in front of it. From head to tail the pancreas measures in the infant about 3·5 ctms., and the antero-posterior diameter of the head is a little over 1 ctm.

The *Bladder* is practically entirely an abdominal organ at birth; but I shall not consider it at this time, as I have already done so fully in a paper entitled "The Pelvic Viscera in the Infant," which appeared in the *Edinburgh Medical Journal* for August 1890.

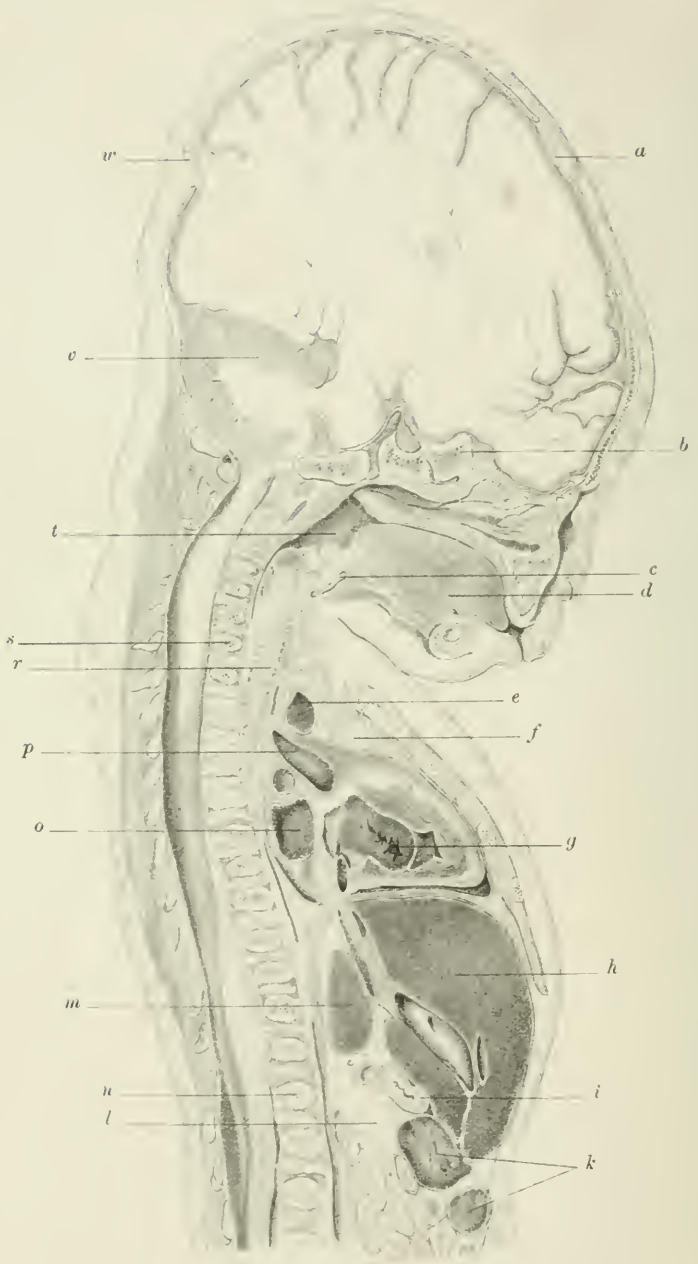
The following important points in this communication may be briefly re-stated as follow:—

1. The relations of the abdominal viscera, liver, stomach, spleen, etc., to each other and to the bony landmarks, and more especially the vertebral column, have been pointed out, so as to lay a sure foundation for their clinical investigation by means of palpation and percussion.

2. The form and dimensions of the liver, stomach, spleen, and supra-renal capsules have been stated, so as to throw some light upon certain peculiarities in the infantile economy, *e.g.*, the ease with which infants vomit, the common occurrence of prolapsus recti.

3. The frequency of an abnormal disposition of the large intestine, especially of the sigmoid flexure, has been insisted upon as giving a clue to the cause of some cases of obstinate constipation in infants, and as affording a warning to surgeons who may have to open the abdomen for imperforate anus, etc.; and

4. The whole subject has been investigated by the frozen sectional method, so as to insure the least possible disturbance of relations.



Mesial Sagittal Section of New-born Infant ($\frac{5}{8}$ *Natural Size*).

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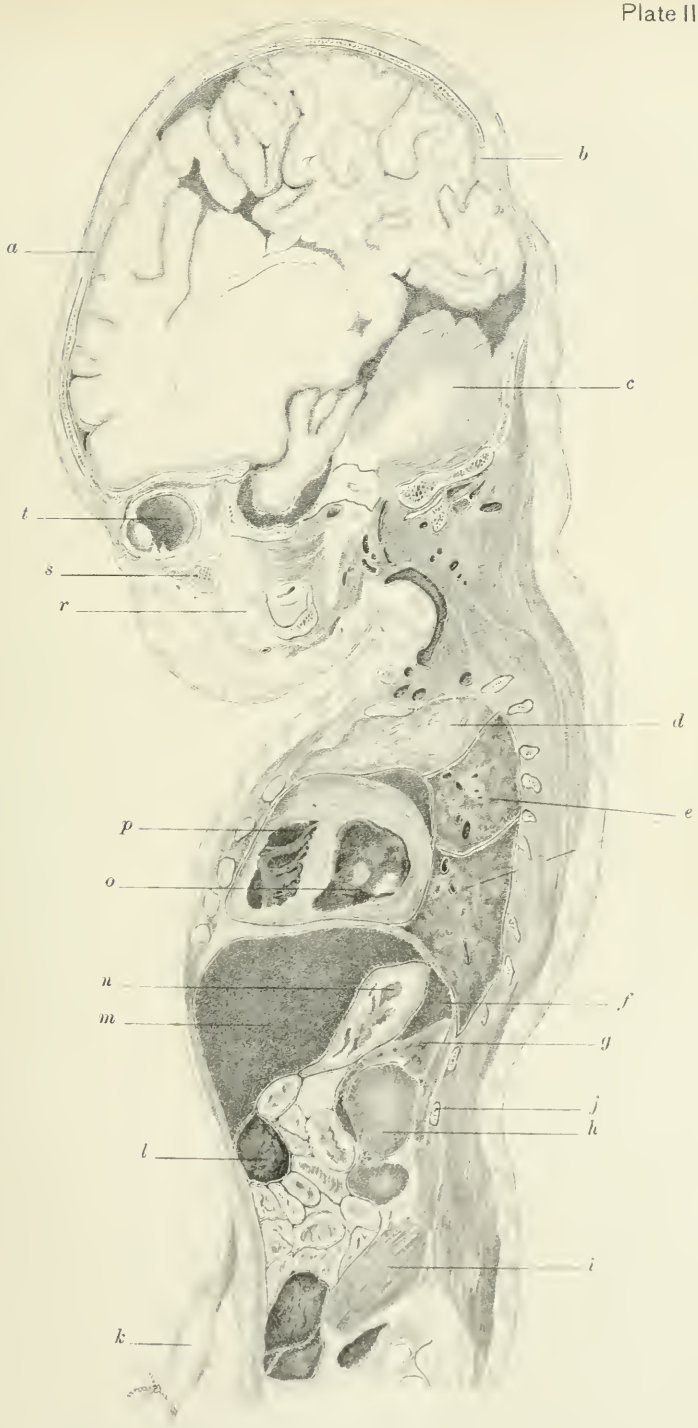

Left Lateral Sagittal Section of New-born Infant ($\frac{1}{3}$ *Natural Size*).

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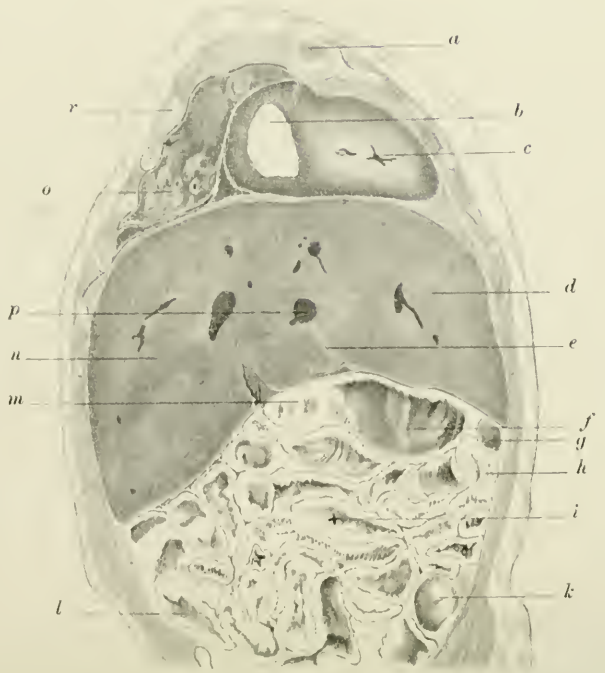
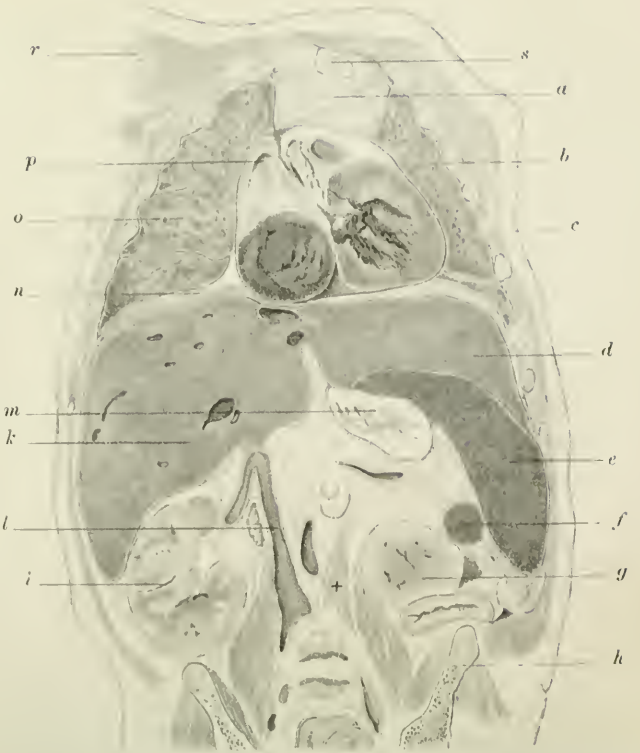
Fig 1



Coronal Section of Thorax and Abdomen
of New-born Infant (§ *Natural Size*).

Fig 2



Coronal Section of New-born Infant, Thorax and Abdomen,
posterior to Fig. 1 (§ *Natural Size*).

Plate III

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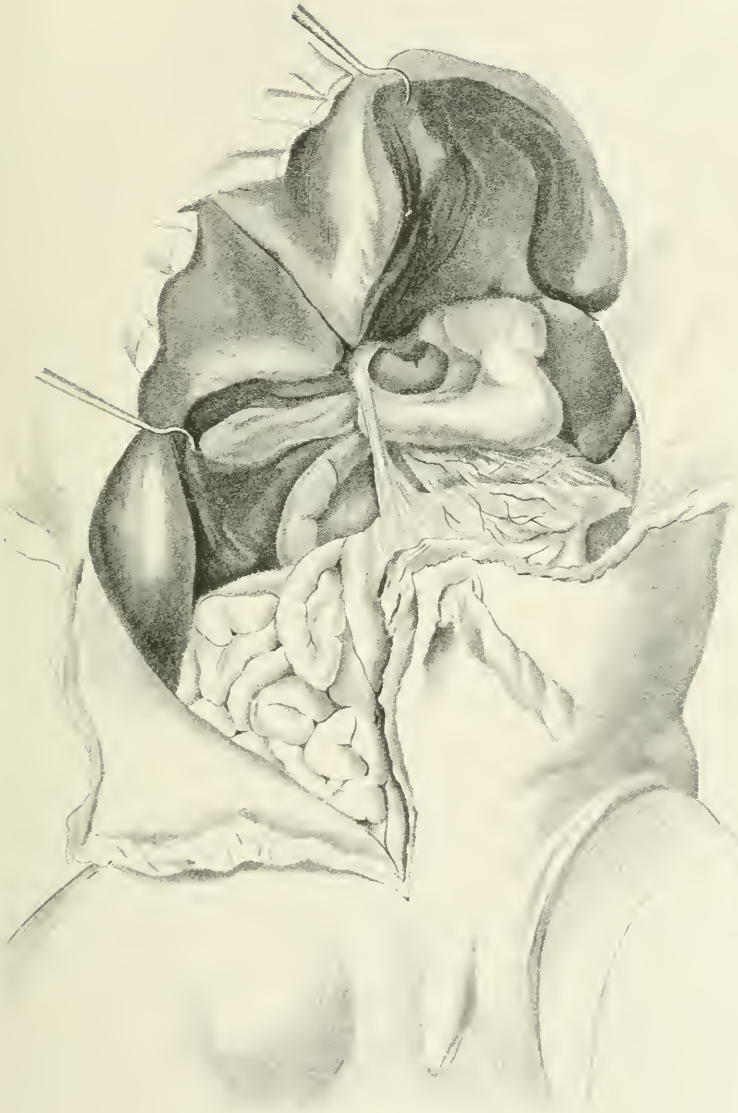

Dissectional View of Abdominal Viscera in New-born Infant (⅔ *Natural Size*)

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Description of Plates.

PLATE I.—Mesial Sagittal Section of New-born Infant. In the upper part of the region of the Head the section has passed slightly to the right of the middle line, leaving the falx cerebri unexposed. *a*, Anterior Fontanelle; *b*, Presphenoid; *c*, Thyroid Cartilage of Larynx; *d*, Tongue; *e*, Left Innominate Vein; *f*, Thymus Gland; *g*, Tricuspid Opening in the Heart; *h*, The Liver; *i*, The Pylorus; *k*, Transverse Colon; *l*, The Pancreas; *m*, Lobus Spigelii of Liver; *n*, First Lumbar Vertebra; *o*, Left Auricle; *p*, Aorta; *r*, Trachea; *s*, Seventh Cervical Vertebra; *t*, The Pharynx; *v*, Cerebellum; *w*, Posterior Fontanelle.

PLATE II.—Left Lateral Vertical Section of New-born Infant. Right face of Section seen, $\frac{5}{8}$ natural size. *a*, Anterior Fontanelle; *b*, Posterior Fontanelle; *c*, Cerebellum; *d*, Thymus Gland; *e*, Left Lung, Upper and Lower Lobes; *f*, Spleen; *g*, Left Supra-renal Capsule; *h*, Left Kidney; *i*, Left Psoas Muscle; *j*, Twelfth Rib; *k*, Umbilical Cord; *l*, Transverse Colon; *m*, Liver, Left Lobe; *n*, Stomach; *o*, Left Ventricle of Heart; *p*, Right Ventricle of Heart; *r*, Left Sucking-pad; *s*, Left Malar Bone; *t*, Left Eye.

PLATE III.—Coronal Sections of Thorax and Abdomen of New-born Infant, $\frac{5}{8}$ natural size.
Fig. 1.—Coronal Section of Thorax and Abdomen in Plane of Stomach. *a*, Sternum, Third Ossific Centre; *b*, Clot in Right Auricle of Heart; *c*, Right Ventricle of Heart; *d*, Left Lobe of Liver; *e*, Longitudinal Fissure of Liver; *f*, Stomach; *g*, Spleen; *h*, Transverse Colon; *i*, Position of Umbilicus; *k*, Sigmoid Flexure; *l*, Cæcum; *m*, Duodenum; *n*, Right Lobe of Liver; *o*, Right Lung, Middle Lobe; *p*, Umbilical Vein; *r*, Fourth Costal Cartilage (right).

Fig. 2.—Coronal Section of Thorax and Abdomen in plane posterior to that in Fig. 1. *a*, Thymus Gland; *b*, Left Lung, Upper Lobe; *c*, Left Ventricle of Heart; *d*, Left Lobe of Liver; *e*, Hypertrophied Spleen; *f*, Accessory Spleen; *g*, Left Kidney; *h*, Left Iliac Crest; *i*, Right Kidney; *l*, Vena Cava Inferior; *k*, Right Lobe of Liver; *m*, Stomach near Fundus; *n*, Diaphragm; *o*, Right Lung, Middle Lobe; *p*, Opening of Superior Vena Cava; *r*, Coracoid Process; *s*, Manubrium Sterni.

PLATE IV.—Dissectional View of Abdominal Viscera in New-born Infant, $\frac{3}{8}$ natural size. The liver has been drawn upwards in order to show the position and form of the stomach. The transparent character of the great omentum is well displayed, and the green colour of the meconium in the transverse colon can be seen shining through the omentum. The umbilical vein is shown passing upwards in the falciform ligament of the liver.

Dr Symington remarked that the peculiarities in the relations of the abdominal viscera of the newly-born child were mainly due to the large size of the liver, and the fact that the small intestines were usually nearly empty, while the large were distended with meconium. With reference to the liver, he agreed with Dr Ballantyne in considering that it might be described as possessing six surfaces instead of five, as in the adult. The additional surface was a left one, as a considerable surface of the liver comes in contact with the left lateral wall of the abdomen. He would like to have Dr Ballantyne's opinion with regard to the relations of the peritoneum to the descending colon. From his own observations he was inclined to believe that Mr Treves had exaggerated the frequency of a descending meso-colon.

Dr Lockhart Gillespie, wishing last summer to discover the relations and position of the stomach in infants, made a frozen section of the body of a child six days old. He excised a wedge-shaped piece of the thorax and abdomen, the apex of which opened into the stomach from the pylorus nearly to the top of the fundus. The organ was dilated and full of milk and gas; it lay with its long axis almost parallel to the long axis of the body, with only a slight curvature to the left. He took exception to Dr Ballantyne's drawing of the natural position of the stomach, for he did not think that it was ever bent on itself at such an acute angle. When dilated even it seemed to be only slightly curved, and it would be interest

ing to know whether this was due to a rising of the fundus, or to a greater mobility of the pylorus in infants as compared with adults.

Dr Wm. Russell expressed the pleasure with which he had listened to Dr Ballantyne's paper, and considered any good work on the relations of the viscera to the surface of the body of great value. When Dr Symington's work on the *Anatomy of the Child* was published he had perused it with much profit, and Dr Ballantyne's work was in the same direction. He thought that if more attention were paid by clinicians to the relations of the organs to the surface, some curious mistakes would not so frequently be made as they are. Referring to one of Dr Ballantyne's figures as to the shape and position of the stomach in the fœtus, he said that he had seen it the same shape and lying in the same position in obstruction at the cardiac end, and also in one case at least of marasmus in chronic phthisis.

Dr Church—considering the manner in which the stomach of the child is pressed upon by neighbouring organs—suggested that more attention ought to be paid to the posture in which the child is laid as a means of relieving or preventing vomiting. He also thought that constipation might to some extent be prevented by bearing in mind the points in which the large bowel varied in many children from that of the adult, and posturing the child accordingly. The finding of meconium in the stomach was interesting as showing, from whatever cause, a reversed peristaltic action of the bowels in the case referred to.

Dr Ballantyne was glad to find that Dr Symington supported him in the conclusions at which he had arrived, and he took this opportunity of stating that it was from the study of Dr Symington's monumental work on *The Anatomy of the Child* that he (Dr Ballantyne) had first been led to investigate the topographical anatomy of the infant. Dr Ballantyne was glad to be able to confirm Dr Symington's remarks concerning the arrangement of the peritoneum in relation to the descending colon; he had in no case found the slightest trace of a mesentery. In reply to Dr Gillespie, it was of course possible that some slight displacement of the stomach might have taken place in the specimen he had referred to; it was a simple dissection and not a frozen section, and was liable to fallacy. A good coronal section passing slightly obliquely and in the axis of the stomach was still a desideratum. In reply to Dr Church, he thought that the presence of meconium in the stomach in one case was due at least in part to the mechanical compression to which the body of the child had been subjected in labour. He had to thank the members for the kind and attentive hearing they had given to his paper.

Meeting VIII.—May 6, 1891.

Professor A. R. Simpson, *President, in the Chair.*

I. Exhibition of Patient.

Dr John Thomson showed a case of Addison's keloid on the right hand of a male infant aged 13 months. The patient was perfectly healthy in all other respects, and was the only child of perfectly healthy parents. Six months ago the mother noticed a small wart-like lump on the radial side of the little finger, on the right hand, near the terminal joint. There had been no abrasion of the skin nor any injury whatever. It caused no pain, and little attention was paid to it, until some months later it was seen to have spread and to have caused flexion of the finger. The flexion of the finger increased steadily, and other similar nodules appeared on it; and three months ago the adjacent aspect of the ring-finger became implicated. One month ago, a similar nodule was found on the skin over the ulnar margin of the hand, and now there were two in that position. The disease consisted of hard nodules connected by bands of hard scar-like tissue, which, although originating in the skin, affected the entire thickness of the soft parts. There had evidently been considerable contraction of the morbid tissue, as the fingers were fixed in a flexed position and had their terminal phalanges inclined laterally toward one another. No cause of any kind could be discovered. The distribution suggested the probability of nervous influence. Arsenic had been given for three weeks, but the disease had increased in prominence distinctly during that time.

II. Exhibition of Specimen.

Dr Affleck showed a case of abscess of the liver.

Prof. Grainger Stewart stated that he had the patient under his care in the summer and autumn of 1889. That she then had enlargement of the liver; and that he, suspecting hydatid or abscess, carefully explored the liver with an aspirator. On the first occasion he got off a little fluid, about a drachm, stained with bile and without hooklets, nor any marked amount of chloride of sodium. On the second occasion nothing was obtained. In these circumstances, and considering the irregular lumpy enlargement of the organ, it seemed most probable that syphilis was the cause. She was put upon anti-syphilitic remedies, and improved markedly,—the liver diminishing in size, uneasiness disappearing, and her general health becoming gradually better. She left the hospital some time afterwards and got married, and did not again come under Prof. Stewart's observation.

III. Original Communications.

1. FŒTID PLEURAL EFFUSION.

By Alexander James, M.D., F.R.C.P. Ed., Assistant Physician, Royal Infirmary, Edinburgh; Lecturer on the Practice of Physic, School of Medicine, Edinburgh.

Under this head are comprised—(1), Effusions, fœtid or stinking, because associated with, and resulting from, gangrene of the lung and superjacent pleura; (2), Effusions which, as the result of the access of air or of putrid materials, have become fœtid; and (3), Effusions which are fœtid, but in which by symptoms, by post-mortem examination, or even by both combined, the existence, on the one hand, of gangrene of the lung, and on the other of the means of access of air or putrid material, cannot with certainty be demonstrated.

Gangrenous Effusions.—The most cogent illustrations of these are the instances in which the dependence of the affection on lung gangrene is demonstrated by the discharge of necrosed pieces of lung tissue through the chest wall opening, after paracentesis. Of such the following *résumés* are probably the best examples:—

(Wagner, *Berlin. Klin. Woch.*, Sept. 6th, 1880.)—A lad, aged 17, was admitted to hospital Oct. 14th, 1878, suffering from dysentery. For this he was treated with salicylic water injections, and in about two weeks he had quite recovered. On Nov. 6th, however, he became again ill with symptoms which looked like those of an acute pneumonia of the left lower lobe, viz.,—rigors, pain in the side, cough, and rusty, viscid spit; pulse, 120; temp., 40° C. (104° F.) On the eighth day of this pneumonia the temperature began to fall gradually, and the lysis was complete on the twenty-first, the percussion dulness also getting gradually less. After the temperature had been normal for three days he was again seized with pain in the left side, and pleurisy with effusion was diagnosed. On the twenty-fifth day an exploratory puncture revealed the presence of a white, thinnish pus of a disagreeable odour, and showing by the microscope bacteria. Paracentesis was performed, and 700 grammes of a disagreeable but not quite putrid character were removed. For three days after this a discharge continued, but on the third day a piece of gangrenous lung tissue 7 cm. by 3 cm. ($2\frac{1}{4}$ in. by nearly 1 in.) was removed through the wound opening. All the symptoms improved after this, but during the healing of the wound it was noticed that on washing out the pleural cavity with carbolic solution some of the fluid was expectorated, indicating the existence of a communication between this cavity and the bronchus. By Feb. 16th the wound had healed, and a yellow spit was all that remained to tell of the lung trouble. This by June 16th had disappeared, leaving the patient quite well.

The explanation of these events Wagner believes to have been that from the dysenteric bowel an infective process of embolic nature set up the pneumonia, and that this was followed by lung gangrene. He also considers that the fact that the fluid in the pleural cavity was at first not very fœtid was due to the gangrenous part of the lung being for a time covered in by the pleura and layer of inflammatory false membranes, so that only a fractional amount of putrid material obtained access to the effusion. The subsequent communication with a bronchial tube and consequent expectoration of carbolic solution was, he believes, due to the corroding action of the fluid on the cicatrizing portion of lung surface.

A very similar case has been reported by Ewart (*British Medical Journal*, vol. i., 1887).—A boy, aged ten years, while convalescing during the fourth week of typhoid fever, developed symptoms which looked like those of peritonitis, but which shortly indicated left-sided pleurisy with effusion. After a week he was aspirated, and ¾ pint of thick shreddy pus was removed, with relief to the most urgent symptoms. The following day the chest was opened freely in the anterior axillary line (fifth space) and in the scapular (ninth space), a large quantity of pus escaping. Free discharge continued, but no injection was used. On June 15th, perflation was performed under the spray in the manner depicted in the *Lancet*, July 31st, 1886. The air was delivered into the centre of the chest through the anterior opening and allowed to escape only at the posterior. The result was the expulsion of fœtid pus, of a piece of necrosed lung, and of heavy false membranes. A smaller piece of false membrane was expelled by perflation the next morning, and a small piece on the third day. From this time the fœtor ceased and the amount of pus decreased rapidly. On the eighth day the discharge was turbid and serous, and it remained serous to the end. Both wounds were closed on the thirtieth day from the date of the incision.

In other cases the dependence of the effusion on gangrene of the lung is indicated only by fœtid expectoration. Of such the following is a good example (Frænkel, *Berl. Klin. Woch.*, April 28th, 1879).

A boy, aged 12 years, fairly well built, was suddenly seized with symptoms indicating pleuro-pneumonia of the right lower lobe. The crisis occurred on the eighth day, but during the next twenty-four hours the temperature began again to rise, and the other symptoms—pain, dyspnœa, etc.—to return, with physical signs of increasing effusion. On the fourteenth day after the crisis fœtid expectoration suddenly occurred. Lung gangrene was accordingly diagnosed, paracentesis was performed, and 500 cc. of fœtid fluid were evacuated. In two months the patient was quite recovered. In this case the portion of lung which had become gangrenous was probably so small that its separation in broken-down granular and molecular matter and not in mass was possible.

In other cases neither fœtid expectoration nor the discharge of gangrenous portions of lung through the chest wall has occurred. Thus, J. B., age 53, a domestic servant, was first seen by a doctor April 3rd, 1887. She stated that she had been exposed to cold eight days previously; that on the evening of that day she had had a shivering, and that ever since she had felt very ill. She had great pain in the left side on taking a long breath and on coughing, and she stated that this pain and cough had been getting so much worse, and that she had been getting so weak that she was now compelled to remain in bed. Finding some impairment of the percussion note about the inferior angle of the left scapula, with weakening of the breath sounds and slight crepitation, a pleuro-pneumonia was diagnosed, and she was sent to hospital. On admission her temperature was 103°·2 F.; pulse 120, very soft and compressible; respirations, 25 per minute. She had cough with scanty mucous expectoration, severe pain in the left lower and lateral region of the chest, and the physical signs indicated pleural effusion, the dulness reaching nearly as high as the spine of the scapula posteriorly and the fifth rib in the axillary line. On the second day after her admission the pain was somewhat easier, but with the dyspnœa, fever, and sweatings, her general condition was manifestly worse. An exploratory puncture was made, and a hypodermic syringeful of grayish-yellow watery and horribly fœtid pus containing bacteria was drawn off. On the same day the chest was freely opened and about 60 oz. of a similar fluid was evacuated. Little improvement followed, and the patient rapidly becoming more collapsed, death occurred on the evening of the following day.

On post-mortem examination the upper part of the left lung was found adhering to the chest wall. The lower part was collapsed and œdematous, and its surface was covered by a layer of grayish-yellow false membrane. On section there was found on the posterior surface of the lower lobe a patch of gangrene, shaped like a very flat triangle, the base of the triangle corresponding to the pleural membrane, and being in extent from above downwards about 2 inches. The aorta was atheromatous, and the heart distinctly fatty. In this case no fœtid spit had been detected, but of course it might be maintained that had a better-nourished heart enabled the patient to live a little longer the characteristic fœtor would have been met with.

But now, if two such clear indications of lung gangrene as fœtid spit and the discharge of sphacelated lung tissue be not present, how are we to be certain in any case of stinking effusion which recovers, or in which no post-mortem examination is obtainable, that we have been dealing with an effusion due to lung gangrene? This can, however, best be understood after considering the subject of putrid effusions.

Putrid Effusions are effusions in which decomposition has taken

place as the result of the access of putrefactive organisms. This access may be brought about in very many ways. It may be directly through the chest wall as the result of repeated tappings when antiseptic precautions have not been sufficiently attended to. Although nowadays such cases are but seldom met with, still they occur, the usual sequence being a sero-fibrinous effusion treated by repeated tappings becoming gradually purulent, and this, treated by free incision, becoming in turn fœtid. But it may occur also in a less easily avoidable manner, *e.g.*, an empyema may have opened through the chest wall before being seen, or it may have opened into a bronchial tube, and through the opening by which the pus escapes, air with its contained germs may enter. Again, as the result of the rupture of a lung hydatid or abscess, putrid effusion may occur; whilst a phthisical cavity situated superficially may burst into the pleura, producing pneumothorax, and the sero-purulent effusion which in time shows itself may be fœtid. As a fact, however, the exudation in cases of tubercular pneumothorax has little tendency to putrefy as compared with that of all non-tubercular cases in which an opening is established between the bronchi and the pleural sac. This has been explained on the theory that tubercular fluids do not form a suitable soil for the growth of other micro-organisms (*Berl. Klin. Wochenschr.*, No. 20, 1888), a theory for and against which, however, something may be said. Thus it is to be remembered that the putrefactive changes brought about in tubercular sputum as the result of long keeping do not seem to diminish the potency of its contained tubercle bacilli, and that surgeons tell us that septic and putrefactive organisms in a wound favour rather than impair the power for harm of a tubercular process. Be this as it may, I have no hesitation in expressing the opinion that this antagonism theory may to a considerable extent be a correct explanation. Fœtid lung cavities rarely become tubercular; and among the few cases of tubercular phthisis which I have seen in which, after the formation of cavities, a fœtid spit and fœtid odour of breath have shown themselves, I have witnessed some very striking instances of amelioration in the symptoms, physical signs, and general condition of the patients.

Other causes of putrid effusions are ruptures into the pleural cavity of a gullet as the result of malignant disease, and ruptures into the pleural cavity from the abdominal. Thus a gastric ulcer perforating the diaphragm may act in this way.

Remembering that abscesses beneath the diaphragm, like all collections of pus in the abdominal cavity, are specially apt to be fœtid, and remembering also that abscesses beneath the diaphragm are likely to entail inflammatory and suppurative changes above it, *i.e.*, in the pleural cavity, either by perforation of that structure or by absorption processes through it, we should be prepared to meet with putrid empyemas as frequent associates of sub-diaphragmatic abscesses. This we do, but I am persuaded not so frequently as we

might expect. Thus in a case in Ward 23 (M'Peet), in which an abscess of the spleen had caused an empyema of the left side, there was fœtor, neither of the abdominal nor of the pleural pus; and in another case in Ward 23 (Tyler), in which, with tubercular peritonitis and waxy spleen, a large collection of pus beneath the diaphragm had given rise without perforation to a right empyema, although the pus underneath the diaphragm was decidedly fœtid, that above was not so.

But now, just as we have noted at page 160, that difficulty must occur in connexion with the diagnosis of gangrenous effusion, should fœtid spit be absent and should there be no discharge of sphacelated lung tissue, so a difficulty must occur in connexion with the diagnosis of putrid effusion in cases where means of access of putrid germs to the effusions are not evident.

This leads to the consideration of the third class of fœtid effusions, namely,—

Effusions which are Fœtid, but in which by symptoms, by post-mortem examination, or even by both combined, the existence on the one hand of gangrene of the lung, and on the other of means of access of air or putrid material, cannot with certainty be demonstrated.

The following cases appear to be good examples of this :—

(Bouveret, *L'Empyème*, p. 500.) *Résumé.*—A weakly lad of 20 had on Oct. 16th chills and fever, with pain in the right side, cough, and sero-mucous spit, and he entered hospital with these symptoms on the fifth day of the disease, when right-sided pleurisy with effusion was diagnosed. On Nov. 2nd he was distinctly worse, pulse small and frequent, tongue dry, and some œdema over the right chest was noted. Aspiration was performed, and without emptying the pleural cavity 600 grms. of a chocolate-coloured horribly fœtid fluid were drawn off. Little or no improvement followed, and on Nov. 5th a new aspiration withdrew 500 grms. of a similar fluid. The chest was then washed out with a weak solution of phenic acid, a quantity of air entering the cavity during this latter operation. Again no improvement followed, and the patient died on Nov. 7th.

Autopsy.—"Complete collapse of the right lung. The lung shows neither tubercle nor gangrenous foci—it disappears under a thick coating of grayish false membranes. No gangrenous plaques on its surface. Insufflation and careful examination of the organ discovered no perforation. The pleural cavity is filled with about a litre and a half of a brown-green liquid. In that liquid is found a brownish mass, in colour, appearance, and consistence resembling fæcal matter. The weight of this detritus is about 200 grms. (about 6 oz. Troy). It contains numerous leucocytes and a feutrage of fibrils which resist acetic acid. One can ask if these fibrils are not of vegetable nature. M. Malassez considers rather that they consist of long crystalline needles." The other organs in this case were comparatively healthy.

Here, then, is an instance which but for the post-mortem examination would have been classed under the gangrenous pleurisies. It is, however, considered by Bouveret to be an example of putrefactive decomposition of a pleural effusion. As to how the putrefactive germs gained access, Bouveret says, "Nous sommes réduits à présumer qu'ils ont été amenés dans la pleure par la circulation sanguine."

The following is a very similar case:—

J. V., æt. 58, was admitted to Ward 23, July 4th, 1888. His family and personal history were good, and he stated that he had been exposed to cold ten days previously. He had a rigor on that day, and felt very unwell. Next morning he had pain in his left side, which was very severe, and caught his breath. He remained in bed, and was being treated for pleurisy of the left side, but finding no improvement he came to the Infirmary. On admission his pulse was 120, regular and soft; his temperature varied between 99° and 102°; his respirations were rapid and shallow. Examination showed dulness over the left chest, beginning in front at the fifth rib, and in the axillary line at the fourth rib. Posteriorly percussion impairment began about the spine of the scapula, becoming more marked on passing downwards. Over the dull area the respiratory sounds were weak, and at the third, fourth, and fifth spaces anteriorly friction was recognised.

The distress continuing, with shiverings, sweatings, and some wandering at night, and the signs of effusion becoming more marked, the chest was punctured on July 20th. The presence of fœtid pus was in this way clearly demonstrated, but in spite of the removal of this by drainage, he died on July 26th. Fœtid spit was never present.

Post-mortem Examination.—"On the left side a loculated empyema occupying the posterior half of the lower fourth of the upper and the upper three-fourths of the lower lobe. The pleuræ over the remaining portion of the lung united by recent fibrinous adhesions. The empyema did not quite reach the posterior border of the lung at its lower extremity, about three inches of lung being uninvolved in it. The loculus was full of dark gray intensely fœtid fluid, and the pleura covered by a grayish-yellow irregularly eroded membrane. On section, the upper half of the lung was congested, the lower lobe carnified."

But if an exact diagnosis is difficult to arrive at in cases of fœtid effusions in which neither fœtid spit nor the discharge of sloughed lung tissue are met with during life, and in which even by a post-mortem examination neither traces of lung gangrene nor means of access of putrefactive organisms are revealed, much more difficulty is experienced in coming to a conclusion in cases which recover. Of such the following four are fairly typical examples:—

1. P. M., æt. 34, a well-nourished, stoutly-built man, was admitted into Ward 23, March 21st, 1890. He had a good family and

personal history, except that as a bottler in a brewery he was in the habit of taking more than the ordinary amount of beer daily. His complaints were shortness of breath and pain in the right side, and he dated his illness to a period three weeks previously. At that time he had a shivering, and the pain came on with cough, watery and frothy expectoration, and now and again a little vomiting. Notwithstanding these symptoms he never laid up nor consulted a doctor, but kept at work for a week. Then, however, the symptoms continuing, and the pain becoming worse, he laid up in bed and applied a blister to the side. This relieved the pain somewhat, but the other symptoms, cough and expectoration, continued as before. Profuse sweatings occurred, with loss of appetite, thirst, and feeling of weakness, so he came to the Infirmary. On admission his pulse was 100, regular, but slightly dicrotic; temperature, 102°·4; respirations, about 30 per minute. Physical examination of the chest showed a somewhat tympanitic note over the upper part of the right side anteriorly and posteriorly, and over the lower part marked dulness, the upper limit of which was marked out by a line extending from the spine of the scapula posteriorly to the fourth rib in the axillary line, the fourth rib in the nipple line, and the fifth rib in the parasternal line. Over this dull area there was more or less distinct absence of vocal fremitus, respiratory murmur, and vocal resonance, whilst displacement of the heart to the left and of the liver downwards was recognised.

Pleuritic effusion was diagnosed, but as the pulse kept about 110, the temperature varied between 98° and 102°, and as with those changes in temperature profuse sweatings were associated, the probability of purulent effusion was recognised, and an exploratory puncture with the hypodermic needle determined upon. This was performed on March 25th, and after several punctures over the dull area, one in the posterior axillary line resulted in the obtaining of a few drops of a dirty greenish yellow, apparently fœtid fluid. Paracentesis was therefore resolved upon, but as the precise position of the fluid had not been made quite clear by the punctures, and as it was considered possible that the hypodermic needle might be too small to allow the fluid to pass, a small probe-pointed trocar was used instead. To avoid risk of accidents this was connected with a rubber tube, the end of which was immersed in boracic solution. A puncture made at the level of the eighth space in the scapular line gave exit to 40 ounces of fœtid, somewhat shreddy, grayish-yellow pus. Next day, March 27th, paracentesis was performed by Mr Caird, about one inch of the eighth rib at the site of the puncture being resected. A large quantity of fœtid fluid was removed, and a drainage-tube being introduced, the wound was dressed in the usual way with salicylic wool. After the operation the patient expressed himself as feeling better, but the temperature kept varying between 102° and 99°, the pulse about 120, the respirations about 30, and the discharge continued

fœtid. About the 6th April, however, the temperature began gradually to fall, and although on two occasions afterwards it rose temporarily, apparently as the result of retention of some of the scanty fœtid secretion, his progress towards recovery was continuous. To avoid any risk of detention of discharge, the tube, although much shortened, was kept in until June 1st. He was discharged from the hospital on June 18th with the wound nearly healed. Physical examination of the chest then showed a circular patch of dulness about 3 inches in diameter in the region of the operation wound, the breath signs, however, being fairly well heard all over the side.

In this case, although carefully looked for, at no time was the expectoration fœtid, and no discharge of sloughed pieces of lung tissue occurred.

2. (Dr William Russell, *Glasgow Medical Journal*, 1878.) A girl, æt. 21, was first seen Sept. 1st, 1881, with symptoms of left-sided pleurisy. By 8th Sept. the fluid had collected in such quantity that tapping was considered necessary, and about a pint of excessively fœtid pus was withdrawn. Next day the chest was incised, thoroughly washed out with carbolic solution, and a large drainage-tube inserted. In a few days the discharge became sweet and less in quantity, but for a time a cough was troublesome. On Oct. 3rd it was noted that a bad smell was perceptible in her breath after paroxysms of coughing, and on Oct. 13th, as the result of such a paroxysm, she is stated to have brought up about half a teacupful of "what tasted like rotten eggs." After this the cough became much easier, and though it and the fœtor of breath continued for some months, it gradually disappeared, and the patient progressed towards complete recovery.

In this case Dr Russell gives it as his opinion that the pleurisy was not due to lung gangrene, and quoting seven somewhat similar cases, in all of which the left was the affected side, he expresses his belief that the fœtid character of the fluid might be accounted for by the fact of the large intestine being in contact with the under surface of the diaphragm, and consequently in close proximity to the pleura. Further, as regards the fœtid expectoration, Dr Russell gives the following explanation:—

"The presence of a purulent and stinking expectoration on several occasions after the drainage-tube had been withdrawn, and preceded by severe and paroxysmal cough, pointed to a cavity which must have been formed during the healing of the large one. This smaller cavity must, I think, from the clinical history of the case, be regarded as a pleural one, which, becoming over-distended, led to irritation of the adjoining portion of lung, thus causing cough, and finally spontaneous cure by perforation. If we remember how favourable the physical conditions between the diaphragm and the base of the lung are for an islet of infection to become surrounded and shut in by adhesions, which constitute the cure of

such a case, we have a ready and sufficient explanation of this accidental element in the case."

3. (Dr Douglas Powell, *Trans. International Congress*, vol. ii. p. 143.) A young lady of consumptive family had six weeks previously been seized with symptoms of acute pleurisy, which appeared to subside, but recently hectic symptoms had developed, with aphthous mouth and complete intolerance of food by the stomach. The chest signs were those of a large effusion into the left pleura, displacing the heart to the right of the sternum. Paracentesis was advised, and on the second day Dr Stamford removed by the aspirator 25 oz. of fœtid, purulent matter. Later on the chest was opened and a tube introduced, and at last accounts the patient was doing well.

4. A. K., æt. 31, joiner, was admitted to Ward 23, April 3rd, 1891, with symptoms and signs of pleuritic effusion. His family and personal history were good; his surroundings at home and at work were quite equal to the average. He had been a sergeant in the artillery, and had served for ten years, and he had always been fairly temperate. His height was 5 ft. 9½ in., and his ordinary weight was about 12 stones.

Present illness dated from about three months before his admission, when he stated that after having noticed himself somewhat out of sorts for a week, he was, after some exposure to cold at work, seized with pain in the back, which was very severe and continued for days. This gradually mended, and after some three weeks in bed he began to move about. Cough then began to affect him; but in spite of this and of the weakness and breathlessness which he was experiencing, he continued to go about and do light work. Gradually, however, his symptoms became aggravated, and on March 28th he had again to take to his bed. He was then advised to come to hospital, and he came and was admitted on April 2nd.

On admission, although his general condition was fairly good, he was evidently pulled down. His weight was 10 st. 6 lbs. Resp., 25; temp. varied between 97° and 100°·5; pulse, 75, regular, rather soft. He had a cough, usually slight and dry; but he had no chest pain unless the cough became troublesome.

Physical examination revealed left-sided pleuritic effusion, and it was noticed that the area of dulness and of absent fremitus, respiratory murmur, and vocal resonance was situated more posteriorly than in ordinary pleural effusions. The "curved line" was distinct, but its highest point was about the spine of the scapula, and percussion from above downwards in the mid-axillary line revealed no dulness whatever—*i.e.*, the anterior limit of the dull area was about the posterior axillary line.

The heart was in its normal position, and the tympanitic note of the stomach in front could be detected a little higher than usual. Tongue was clean; the patient enjoyed very good appetite and digestion, and the bowels were regular.

In this case, owing to the general mildness of the symptoms, I diagnosed pleurisy with sero-fibrinous effusion. The facts, however (1), of the initial pain having been very severe, and (2) of the position of the dull area being situated more posteriorly than usual in such effusions, were noted; but it was believed that the great pain had been due to some muscular rheumatism, or to some associated nephritis, of which there had at first seemed to be some indications, and that the position of the dull area might be explained on the supposition that during the effusion stage he had been lying on his back.

I therefore ordered rest in bed, good diet, and iron and quinine internally, and waited a few days to see if then any indications of absorption would show themselves. On April 9th I examined his chest again, and finding absolutely no change in this respect, I proceeded to aspirate, intending by removing a few ounces of fluid to favour further absorption. To my surprise, however, I found brownish-yellow purulent fluid, with a horribly fœtid odour. The aspiration was at once stopped, and I arranged with Mr Caird to have the operation of free incision and drainage performed next day. Owing mainly to the objection of the patient the operation was postponed, and on the afternoon of April 13th he suddenly coughed up a large quantity of frothy, purulent expectoration, having a somewhat fœtid odour, and showing under the microscope streptococci, diplococci, and some bacilli. This expectoration continued to come up in large quantity all that day and the following night. Next day the operation was performed, the incision being made in the line of the inferior angle of the scapula, and a small portion of the seventh rib being resected. After a large quantity of extremely fœtid brownish-coloured fluid had welled out, a drainage-tube was inserted, and the wound dressed as usual with salicylic wool. The subsequent course has been very satisfactory. The expectoration ceased within a few days of the operation, the discharge has gradually diminished and lost its fœtor, and the lung has expanded. At no time has any piece of gangrenous lung tissue come away.

And now, How can the presence of putrid effusion in such cases be explained? Bearing in mind that in one or other of them a small and superficial patch of gangrene of the lung may possibly have been existent without making its presence known by fœtid spit or discharge of expectorated tissue, or that a small superficial lung cavity may possibly have been present, and by its rupture allowed a maximum of germs and a minimum of air to enter the pleural cavity, we are yet bound to admit that these hypotheses are not satisfactory. Remembering, too, that fœtid sub-diaphragmatic abscesses may set up non-fœtid empyemas, it seems difficult indeed to divine how to the pleural fluid in such cases putrid germs have gained access. As a working hypothesis, however, I would suggest one based on observations made many years ago by Mr Caird.

Mr Caird took freshly killed cats, rabbits, and pigeons, and having carefully washed the skin, etc., with antiseptic solutions, he opened the bodies under the spray, and with strict antiseptic precautions inoculated flasks of sterilized meat infusions with portions of liver, lung, kidney, spleen, muscle, and blood. On incubating these he found, after an interval of time, that every one of them contained micro-organisms, and that in the case of the liver these were in some instances putrescent.

There can, I think, be no doubt that normally the various tissues contain micro-organisms, some of them putrefactive; but that owing to the phagocytic action of the normal tissues, their effects for harm are prevented. Given, however, some exhausting condition—as, for example, convalescence from acute disease or chronic alcoholism—this phagocytic action will, of course, be lessened; and to this let an acute pleuritic process be superadded, it is not unreasonable to believe that in connexion with the inflamed tissue these germs may be able to secure for themselves a nidus for their growth and development.

The Etiology and Symptomatology of fœtid pleural effusion are those of pleurisy, empyema, lung gangrene, and the various conditions which lead to the access of putrefactive organisms to the effusion. They, therefore, need not be considered in detail; attention need only be paid to certain peculiarities which, though not absolutely characteristic, are yet of special importance in connexion with the recognition of the malady. Further, as the etiology and symptomatology of the second variety of fœtid effusion, viz., putrid effusion, are comprised in the conditions which lead to the access of the putrefactive organisms, the following remarks apply mainly to the first and third varieties, viz., effusions due to gangrene of the lung and fœtid effusions of uncertain origin.

Etiology.—Fœtid effusions occur in childhood and in early and late adult life. They are more frequent in males than in females; and though they may affect the robust as the result of severe exposure, injury to the chest walls, falls, and excessive muscular strain, etc., they are more frequent in those who are constitutionally weak, or who have been weakened by previous disease. Like gangrene of the lung they are frequent in the alcoholic, they seem to have supervened in some cases on ordinary pneumonias, their occurrence has been repeatedly traced to embolic causes, and they have also been met with in cases where tubercular lung mischief might be suspected.

Symptomatology.—Fœtid pleurisy is usually ushered in by specially severe initial symptoms; the shiverings and rigors are well marked, and the fever high. The chest pain is much complained of in the early stages of the affection, to such an extent that even then physicians may suspect that they have to do with something more than a simple pleurisy. The cough is also troublesome, and in cases of gangrenous effusion the expectoration may at an early stage be

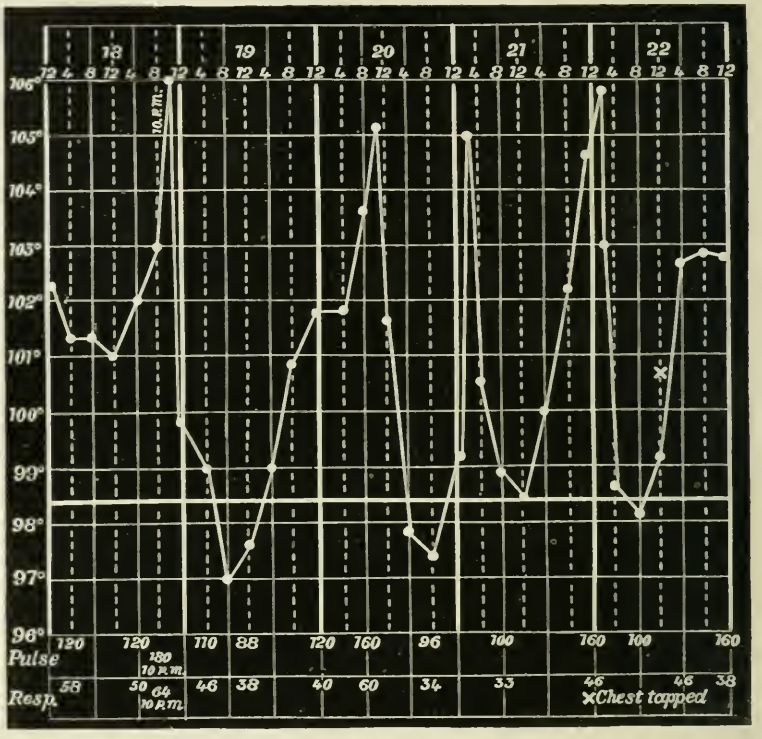
fœtid, and fœtor of the breath may be observed. More frequently, however, those characters of spit and breath are not noticeable till later, and cases occur where they are not present at all. The absence of fœtid spit in a certain proportion of gangrenous effusions is explained as due to the layer of sphacelated lung being in contact only with the smaller bronchi. In this way gangrenous matter cannot readily find its way into the larger tubes to be expectorated, more especially when, let it be remembered, these small bronchi will be rendered practically impermeable by the inflammatory œdema associated with the existence and discharge of the slough. But whilst the absence of fœtid spit does not necessarily exclude gangrene of the lung as the cause of the effusion, it is to be remembered also that the presence of fœtid spit is not necessarily absolute proof of the existence of lung gangrene. As indicated at pages 165 and 166, in cases of fœtid effusions a quantity of the putrid fluid may, by corroding the lung tissue around, find its way into the bronchi, and be expectorated as fœtid spit.

In the course of fœtid effusions the fever soon begins to assume a hectic character, doubtless due to reabsorption of morbid material from the effusion. Large oscillations show themselves in the temperature chart, the rises being preceded by shiverings and the falls by sweatings. The pulse also is apt to become rapid and weak, probably due to the toxic action on the heart of this reabsorbed matter. As the result of the severe chest pain, of the mechanical effects of the effusion, or of the fever, dyspnœa is sooner or later a common symptom of the disease. This condition of fever is well shown in the Chart (see page 170), which is taken from the case of a patient, E., also in Ward 23, two years ago, and in which the fœtid effusion was found by post-mortem examination to have been due to lung gangrene. It is to be remembered, however, that, as in the case of K., page 166, this characteristic fever may be conspicuously absent.

The physical signs are those ordinarily met with in pleural effusions, viz., dulness on percussion, with absence of vocal fremitus, of breath sounds, and of vocal resonance. In cases where the effusion is considerable in quantity these signs are easily discovered over the greater part of the side affected, with displacement of the heart to the opposite side and even of the diaphragm downwards. But with smaller effusions the percussion and auscultation signs are present only over a more limited area, and this area corresponds in the main with the position ordinarily assumed by pleural effusions occurring in patients who have been kept in the recumbent position. That is to say, they will be met with at the lower part of the chest, somewhere between the posterior axillary and scapular lines. Œdema of the chest parietes is a frequent associate of the condition.

By the hypodermic syringe the presence of fluid can be demonstrated and its characters investigated. It has, of course, the characteristic fœtid odour, and though to the naked eye it may present

the appearance of yellow laudable pus, more frequently it is somewhat watery and grayish, or even brownish in colour. Micro-



JAMES E., Ward 23. Fœtid Effusion from Lung Gangrene.

scopically it presents, of course, pus corpuscles with molecular and granular matter, and bacteria, living or dead and immobile. The presence of dead bacteria is interesting, and may readily be explained as being due to the toxic effects on themselves of the products of their own existence. It is possible, too, that the molecular and granular matter may be to some extent the broken-down remains of these organisms thus destroyed.

Course and Prognosis.—Fœtid pleurisy is always a very grave malady; but there is no doubt that, even when it is due to lung gangrene, if the amount of lung tissue affected be not too great, and if proper treatment, viz., free incision and drainage, can be carried out early enough, a remarkably favourable result may ofttimes be obtained. This, too, in cases which appear to have become desperate; and, indeed, writers on this subject expressly state that operative interference should not be neglected, even though the patient is almost moribund.

Treatment.—This is, of course, free incision and drainage at the earliest possible moment. Concerning the operation, the following general points need only be noticed. The point of election for the operation will vary somewhat in each case, and will often be best determined by exploratory puncture with the hypodermic needle or trocar; but, as a rule, it will be in the seventh or eighth interspace, somewhere between the posterior axillary line and the line of the inferior angle of the scapula. This point is in position more posterior than the one usually most suitable for incision in ordinary empyema, or for simple puncture in a sero-fibrinous effusion. It is, however, nearer the ordinary site of lung gangrene—namely, the posterior border—a matter of great importance when it is remembered that in gangrenous cases the portion of lung involved may be large enough to come away as a slough. Further, it is always safer to resect a portion of rib, inasmuch as too early closure of the wound by approximation of the ribs is avoided, and an opening large enough for the free entrance of the drainage-tube and removal of any slough obtained. After the operation, and at the subsequent dressings, as long as a fœtid discharge continues, the pleura may be gently laved out with some antiseptic; warmed boracic solution suits very well. This washing out, however, may often be omitted, but any appearance of sloughed portions of lung tissue must be looked for, and care must be taken lest here and there fœtid discharge be retained by adhesions forming as the lung expands between the pulmonary and costal pleuræ.

It should be remembered that what is desired is to secure free exit to the fluid, escape of false membranes, and any pieces of sloughed lung tissue, and to prevent any retention by the premature formation of adhesions. In the majority of cases the operation above described will be found sufficient. Another plan recommended is the making of two incisions—an upper, *e.g.*, in the fifth interspace, and a lower, in the space next to the diaphragm—the latter being found by passing a probe within the thorax downwards from the first and higher incision. Ewart has recommended "perflation"—that is, the passing through the pleural cavity of antiseptic air, as described in the *Lancet* for 31st July 1886.

It is noteworthy that after the operation, especially in cases of gangrenous pleurisy, the amelioration in the patient's symptoms—fever, sweatings, cough, etc.—is not usually so marked as after the operation in cases of ordinary empyema. This will be readily understood when it is remembered that, whilst in the latter case the purulent effusion is the main cause of the symptoms, in the former the lung gangrene exists and remains for a longer or shorter period of time after the removal of the fluid.

A further point to which reference is made by Frænkel (*Berl. Klin. Woch.*, May 1879) is that evacuation of the fœtid fluid by an aspirator instead of by a free incision may possibly do harm. Frænkel quotes a case in which this was done, and in which during aspira-

tion, suddenly and with a hissing noise, air became mixed with the fluid, indicating the rupture of a gangrenous cavity in the lung. Two days afterwards the chest was opened, coagulated blood escaped through the wound with the gangrenous fluid, the patient had a severe hæmoptysis, his face became cyanotic, and he died, apparently of suffocation, on the operating table. Frænkel considers that in this case negative pressure produced in the pleural sac as the result of aspiration had caused rupture of a gangrenous lung cavity and an opening into an arterial trunk, which up till that time had been occluded by a thrombus.

Whether this is the correct explanation or not, we can readily understand that aspiration in such cases must entail the risk of separating the sloughed portion violently by the strong negative pressure which it can induce in the pleural cavity, and we can also understand that it can never fully get rid of the fluid. But it is also to be remembered that in gangrenous pleurisy the adhesions between lung and costal pleura must ofttimes be not very firm, and that therefore a more than usual risk of lung collapse must accompany the free ingress of air. A piece of folded lint moistened with some antiseptic solution should always during the operation of opening the chest be held over the wound, so as to act after the manner of a valve—that is to say, to allow the fluid to escape, and at the same time guard against the too ready ingress of air to the pleural cavity.

Mr Caird remarked that he was somewhat surprised that Dr James gave such prominence to pulmonary gangrene as the cause of putrid pleural effusions. He thought the proximity of the liver might be cited as a source of infection, much as in subphrenic abscess. In the course of inoculation experiments some years ago he had found that in certain animals the liver invariably gave rise, on incubation, to an infusion which teemed with organisms and had a foul smell, while other organs under similar conditions were sterile, or at most only contained micrococci. In regard to treatment during operation, he pointed out the dangers of rolling the patient on his sound side, or in moving him greatly where the collection of pus was large, or where it was in communication with a bronchus; and he insisted on the necessity of removing a small portion of rib in such cases for drainage, so as to obviate future trouble should the ribs come together after evacuation. He had found that in putrid empyema recovery often took place in a most astonishing and rapid manner.

Dr William Russell considered that no objection could be taken to Dr James's classification of cases of putrid pleural effusion, but those which were of special interest, and on which Dr James specially dwelt, was that division of cases in which there was no clinical or post-mortem evidence that the putridity was due to lung gangrene. Although this was the case, Dr James, if he

understood him aright, seemed inclined to think that these cases were nevertheless due to localized gangrene; if this contention were correct, it would be necessary to explain what became of the gangrenous tissue; and so far as he had been able to follow the paper, this had not been done satisfactorily. Dr James had referred to a paper published by him (Dr Russell) ten years ago, in which he had been bold enough to advance a theory of the possible cause of the putridity, based upon a limited search into the literature of the subject. That paper had been prompted by the record of a similar case by Professors Gairdner and Buchanan of Glasgow in the *Glasgow Medical Journal*—the case being, in their experience, unique. From that time to the present, no important paper on this subject had come under his notice, and he congratulated Dr James on making such an important contribution to the subject, and would draw special attention to the fact that while ten years ago Professor Gairdner, with his long and varied experience, had only seen one such case, we had quite a group of such cases brought together by Dr James, and which had been under his own observation. Whatever view was held as to the cause of the putridity in these interesting cases was, after all, the purely theoretical side; and, as regards the practical side, no sane man could doubt that incision was the rational treatment.

Mr A. G. Miller had listened with pleasure to Dr James's able paper. In reference to Dr James's explanation of possible septic infection in cases in which there was no gangrene or putrefaction, he wished to suggest possible infection from inhalation of organisms. Mr Miller had met with instances of septic suppuration in closed cavities where the only possible source of infection seemed to be the inhalation of drain gas. Might the same cause not act in putrid empyema? Mr Pridgin Teale had published a book on the subject, pointing out that infection of surgical cases might readily occur from drain gas. Mr Miller objected to Dr James's statement that tubercular fluids do not afford good media for the growth of other organisms. From the surgical point his experience was that tubercular abscesses became readily putrid, and the change was always detrimental.

Dr Alex. James, in replying, thanked the Society for the manner in which they had received his paper, and the various speakers for their suggestions. As regards the causes of putrid effusions in the absence of lung gangrene, and in the absence of any channel by which air could gain access, there was room for great difference of opinion, but he thought that Dr Caird's statements best met the difficulty. As regards the theoretical antagonism between tubercular and putrefactive organisms, he was quite willing to accept the statements of Mr Miller and other surgeons. He thought, however, that there might possibly be something in the theory, for, apart from the fact that the pleural effusions in tubercular pneumothorax are so seldom putrid, he had noticed now and

again in a few cases of phthisis, that when the spit became fœtid, and the breath smelt foul, the spread of the lung mischief seemed to be lessened, and the patient's general condition seemed to be rather improved.

2. THE PATHOLOGY AND TREATMENT OF FURUNCULOSIS.

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The usual treatment of boils is generally owned—and I think, with much reason—to be unsatisfactory. In most cases the treatment can only be classed under the head of "expectant"—a euphonistic *confessio ignorantiæ*. In no sense can it be said to be preventive of local recurrence. I propose in this paper to detail a strikingly simple rule of practice which has yielded such gratifying results as to entitle it to be brought before the profession. I shall, therefore, proceed to briefly describe what, so far as I know, are the current views of the pathology of furunculosis, which will greatly aid me in criticising the present modes of treatment, and in describing the line of treatment I desire to advocate.

Pathology.—An essential characteristic of the whole furuncular process is excessive local action, coupled with deficiency of vital power. The earliest premonition of the condition is the appearance of a papule, which is found to occupy the site of a hair-follicle, the hair in many cases being observed protruding through the papule. This papule is soon surrounded by an area of tissue, into which has been effused a more or less large amount of "coagulable lymph," which raises the papule and its adjacent skin above the general surface. Then the papule vesiculates, and, *pari passu* with this, the effusion extends more and more into the surrounding connective tissue, and the vicinity of the papule speedily assumes a faint pinkish hue, which gradually darkens to a deep red, and finally to a dark purple colour. The effusion of lymph is either sufficiently rapid to strangulate the blood-supply of the inflamed centre, or the organisms (which have been introduced *viâ* the hair-follicle or from the blood-stream) are sufficiently powerful to kill outright that portion of tissue immediately contiguous to the hair-follicle, either by their directly irritant effects, or by their formation of a ferment, from tissue-catalysis. There thus results a sphacelus of varying size, which, from its septicity and mechanical presence, produces more local irritation, which is expressed in an increase of inflammatory exudation and a rapid disintegration of the inflammatory products and tissue-elements in its vicinity. We now have present a central slough, outside which is a collection of pus, which in its turn is walled in by inflammatory effusion of low vitality, and tending to mollify. In fact, an acute circumscribed

abscess has resulted. The slough of areolar tissue is often circular, but more often conical in form, and is familiar to the laity as the "core."

In many cases the condition is arrested prior to sphacelation, and a "blind boil" results. This may be explained by a higher endowment of vitality in the connective tissue and other cells of the part, and a more stable condition (*i.e.*, slower vital functions) of the morphological elements composing the inflammatory effusion. Or, again, by the absence of local irritation sufficiently intense to lead to such rapid lymph-effusion as to choke back the blood-supply. Or, if bacteriologically considered, the cells of the part, *plus* the leucocytes and phagocytes, have been able, by massing their forces at the attacked point, to overcome the pathogenic organisms present, and to prevent their secreting injurious products, from the action of which on the tissues fresh and injurious compounds—alkaloidal or otherwise—might be formed. The bacterial invaders being defeated, the victorious army of leucocytes, *et hoc genus omne*, slowly disperses, leaving a barrier to future invasion by that channel in the shape of residual thickening and cicatrization.

The scar thus formed in a "blind boil" does not produce the same deformity as that formed after the discharge of a slough, nor is the resulting pigmentation so great. It is possible, also, that in a few cases of "blind boils" a very small slough is formed which is reabsorbed; and in this case it has probably been formed by vascular strangulation, and is aseptic. When a slough has been discharged, a cicatrix results which is most disfiguring, and attention is directed to it by the surrounding pigment, which is not reabsorbed for months after.

The pus formed in a furunculus is of an extremely unhealthy character. The late Professor Spence described it as "only an imperfect pus, whose evacuation gives little or no relief; often, indeed, the reverse, on account of the irritation set up by the interference. It consists of portions of broken-down cellular tissue, mixed up with sloughy, unhealthy pus." The "breaking" of the boil is due to the loosening of the "core" by the suppuration of the tissue round about it, and the pressure to the free surface of skin of the pus thus formed.

In two recent cases of mine at Meerut, children suffering from bronchitis, complicated with post-natal atelectasis, the result of the debilitating effect of two hot seasons in the plains, a peculiar character was given to a crop of boils, from which they also suffered. Here the boils did not sphacelate, but became rapidly converted into small sub-acute abscesses, the pus of which was of a much more "laudable" character than that of ordinary furuncular pus. The area of skin, corresponding to the subjacent area of suppuration, was deeply suffused, and the whole had a "boggy" feeling on palpation. Each of the boils, or rather abscesses, was opened by a fine von Graefe knife, and seemed completely well in about

three days. There was scarcely any residual pigmentation, and no visible scar. Mal-oxygenation of the tissues here evidently led to such a state of low vitality that the inflammatory products effused quickly broke down into pus; but to balance against this supposition we have the phenomenally quick healing of the wound and speedy resolution of the whole process.

As to the organismal cause of furunculosis, Berkeley Hill[1] states that some authors attribute the cause to "a parasitic fungoid plant, the mycelium of which, by developing in a gland-cell, thereby causes limited necrosis (the slough) of the tissues in which it grows. The truth of this view is not clearly established." Again, Lowenberg[2] has found specific cocci in the pus from boils of the external auditory meatus. From the pus of ordinary boils I have myself seen cultivated separately micrococcus auranticus, staphylococcus pyogenes albus and aureus (the two latter occur in ordinary pus), and a white torula, as well as several unnamed cocci and bacteria.

There is yet another mode of explaining the starting-point of boils. In cases of indigestion, Bright's disease, diabetes mellitus, and in recovery from exhausting pyrexial conditions, there are new and irritating products circulating in the blood—for example, in indigestion, ptomaines and other chemical compounds, produced by the action of abnormal gastric, hepatic, or intestinal secretions on the food and mucus of the gut; in Bright's disease, urea and its derivatives; in diabetes, glycogen and allied substances; in dyscrasic conditions of the system, compounds produced by incomplete or abnormal tissue metamorphosis. These all being excreted cutaneously, give rise to severe local irritation in the process, and favour the incursions and development of organisms lying on the skin-surface or circulating in the blood-stream, which healthy cutaneous secretions, coupled with a certain standard of vitality in the tissue-cells of the part, may have hitherto held in check. The question is naturally suggested, "Why, then, in chronic diseases, such as Bright's and diabetes, are boils not a constant feature of the disease?" It can only be presumed that the standard of cell vitality in these diseases, though certainly depressed, only occasionally falls below that limit of health by which it is enabled to resist the action of the germs or their products. It is often noticeable that it is towards the end of these diseases that the patient's sufferings are accentuated by an attack of furunculosis. At this period the vitality has reached a sufficiently low ebb to be no longer able to keep the organismal invading host, or other irritant, at bay.

Local irritation is generally only of avail in causing boils when there co-exists at that time the peculiar furuncular dyscrasia. This is a condition which is very imperfectly understood, nor by what it is directly caused.

[1] Quain's *Dictionary of Medicine*, art. "Boils."
[2] *Zeitschrift für Ohrenheilkunde*, x., 1881.

Etiology.—Predisposing Causes.—The male sex, and the period of middle life (B. Hill [1]) or youth (Erichsen [2]), are stated as very specially liable to furunculosis. It must, however, be familiar to most practitioners that there is no such limitation, or even preference. The disease occurs at all ages, and in both sexes, fairly uniformly distributed.

Again, a plethoric habit *per se* is insufficient to cause it, though such a habit generally co-exists with very favourable circumstances for its development, such as overfeeding, or a diet including too much flesh in its composition, or too much nitrogenous food, *plus* a lessening of the power of oxidizing it by over-indulgence in wine, spirits, or malt liquors.

The seasons of autumn (B. Hill),[3] spring, and summer are, in Britain and Germany, supposed to favour the occurrence of furunculosis. The latter certainly has an effect, probably from the additional stimulation of the skin and its glands by the heat, as well as by favouring the life and growth of the specific organism of the disease. In India we find it specially common in (though not entirely confined to) the hot and rainy seasons, *i.e.*, from March to the middle of October. This prevalence is undoubtedly due to the general exhaustion of the frame during these seasons by the great heat, copious sweats, and sleepless nights; and, locally, to the irritation of the exhausted skin and its glands by the accumulation and decomposition of the elements of the sebaceous and sudoriparous secretions. When we recall these—olein, palmatin, cholesterin, albumin, extractives, urea, and formic, acetic, butyric, propionic, capric, and caproic acids—we see ample material for the production, during decomposition, of very irritating products. Eating unripe mangoes is well known in India to occasionally cause boils; this effect, no doubt, is due to the irritation of the cutaneous excretory apparatus by what seems to be a terebinthinate principle in the *unripe* mango. By this irritation, either local resistance to germ invasion is lowered, or sufficient inflammation is directly produced to lead to sphacelation. I do not think that this could occur in the absence of the usual depressing influences at work in the hot season or early part of the rains, when unripe mangoes abound. In the rainy season, especially, the *vis resistentiæ* is lowered by the depressing effects, on the nervous and other systems, of the saturated atmosphere, malarial poisoning, and the sedentary habits enforced by the inclement weather. Lichen tropicus ("prickly heat") may, by irritation, become a crop of boils.

Erasmus Wilson long ago maintained an important law, founded on the continuity and approximate similarity of structure of the investing and lining membranes of the body. This law resolves itself into three expressions:—(1.) Disease affecting a part of a

[1] *Op. cit.* [2] *Science and Art of Surgery*, vol. i., eighth edition.
[3] *Op. cit.*

membrane is liable to spread to the whole; (2.) Disease of the mucous membrane may spread to the skin, and *vice versa;* (3.) Disease of a part of a mucous membrane may become translated to a part of the skin, and *vice versa.* This law, it may be presumed, in no inconsiderable number of cases of tropical furunculosis explains many apparently spontaneous attacks of the malady. The first postulate may account for the simultaneous appearance of boils all over the body, as we occasionally see. The second is a most important point to recognise in the tropics, where the *prima via* is selected as the seat of operations of so many morbid processes. Dyspeptic states and hepatic derangements frequently determine furunculosis. The third heading savours of "metastasis," and is not at all well understood. But I think one should recollect these three expressions of a well-recognised law in the diagnosis of the cause and treatment of boils. Another point to consider is the strong sympathy which exists between the biliary and sweat secretions. Both are equally stimulated by tropical heat, and during the exhaustion of their functions, which sooner or later ensues, both liver and skin secrete vitiated secretions, whose excretion by the skin may supply the necessary devitalization of the cutaneous gland-cells, etc., to favour the incursion of pathogenic germs.

Dissecting-room effluvia, by introducing into the system products which are irritating when excreted by the skin, and which at the same time depress the individual's vitality, also cause crops of boils. The same class of causes, *plus* direct local irritation of the skin by morbid fluids in post-mortem examinations, is another occasional cause. So also is the ingestion of diseased or putrefying butcher's meat, acting as it does by the general depressing effects of a more or less severe ptomaine poisoning, coupled with the local irritation caused by their cutaneous excretion.

The depressing effects of exposure to cold and wet, sleeping on a damp bed, extraordinary bodily fatigue, training,—the fatigue of which is often conjoined with an abrupt change of diet from a mixed to, in many cases, an almost exclusively nitrogenous one,—exposure to sewer gas, sea-bathing suddenly begun after a previously sedentary course of life: all these greatly favour, and often directly produce, boils. Sewer gas may also act as a direct irritant, as the H_2S in it is a direct protoplasmic poison.

Last of the general causes, we come to consider the question of epidemics of furunculosis. I observe an account of such an epidemic was reported by Mr Evan Powell to the Nottingham Medico-Chirurgical Society.[1] It occurred in an asylum, and was apparently introduced into the wards by one man. It is stated that there were no insanitary conditions present likely to produce it, and it was probably due to the large number of very feeble cases in the asylum at the time; and it is believed that it spread by infection, and in a few cases by contagion. I saw one such epidemic

[1] *Brit. Med. Jour.*, Dec. 13, 1890.

during the last hot season in India; but it struck me as being more due to the widespread general debility of the men from the heat, profuse sweats, etc., than to a purely epidemic cause. Contagion certainly played no part in its production.

Of *local causes* the following may be cited:—Such occupations as rag-picking and chimney-sweeping may produce it by the occlusion of the hair follicles (by dust, soot, etc.), causing irritation of the skin glands, conjoined with the general mal-nutrition of many of the followers of these occupations. Blisters, stimulating liniments, and poultices, by, in the case of the two former, direct irritation; and in the case of poultices, by their heat and moisture favouring germ growth, are rare causes. We must also take into account the weakening effect of the particular illness necessitating such applications, as most probably these local irritants *per se* would be insufficient for the production of boils. In some text-books "irritation of the sexual organs" is asserted as a cause. Of this I am sceptical.

Excessive perspiration (unattended by tropical heat), for the reasons before given, is another occasional local cause. A very troublesome variety may be produced from the friction of the nates against a boat seat in rowing, but in this case the concomitant effects of training and excessive local sweating must in many cases be considered. The vertical fold between the nates often suffers from the confining of the sweat and the friction of the sodden skin.

Very frequent bathing, followed by friction with very rough towels or coarse horse-hair gloves, as in a course of hydropathy, has in my experience several times, caused furunculosis in people with exceptionally delicate and irritable skins. The boils about the nape of the neck are often caused by frayed shirt-collars or excessive sweating.

Furunculi of the external auditory meatus are generally observed in dyscrasic or debilitated states of the system. They are cut off, from their situation, from most of the forms of direct irritation which can operate in other situations. They often occur in the course of a general crop of boils all over the body.

As to the part of the body most often affected, it has been stated that the anterior half is more liable than the posterior. There is, however, no such preference to be observed practically. The common sites are the face, axillæ, chest, neck, buttocks, knees, and hands; but the palms and soles are never attacked. This simply means that parts devoid of hair are not liable to be attacked; for the process is essentially a phlegmatous cellulitis, spreading from an inflamed gland in connexion with a hair follicle.

There are, however, many cases in which no cause is apparent or ascribable. In these cases the peculiar dyscrasia of furunculosis is most probably present; for in a healthy individual powerful local irritation alone is insufficient to produce boils. That this

dyscrasic state is present, and is combined with a specific germ action, is shown by—(1.) The severity of the type of the disease, ending, as it does, generally in sphacelation, which ordinary localized inflammations, such as acne, lichen, etc., do not. (2.) That pus in almost all cases is auto-inoculable, whereas "laudable" pus from an acne spot or ordinary abscess does not produce any local reaction—at least in the vast majority of cases. Even septic pus does not, when inoculated on a healthy individual, produce anything more serious than at most a small purulent acne spot.

General Treatment.—The first aim, here as elsewhere, is to discover and remove the exciting cause. Thereafter the general health must be improved by such obvious means as regulation of the diet, especially where the condition is believed to arise from too much nitrogenous food, or in plethora from general overfeeding.

Where the system seems to have absorbed a poison, as in ingestion of bad meat, sewer gas, Bright's disease, diabetes, rheumatism, alcoholism, eliminant treatment by purgatives, diuretics, diaphoretics, combined with a hot-air or water bath, and to observance of moderate and regular exercise, light clothing, and other obvious hygienic rules are indicated. If anæmia coexists, citrate of iron and quinine, and strychnia may be ordered. The tincture of the perchloride of iron may be given advantageously when anæmia, unattended with gastro-intestinal or hepatic derangements, alone is present. It has much the same action as in diphtheria, in which disease many practitioners consider it to have almost specific properties. Where debility and emaciation exist, nourishing food, good hygienic surroundings, and cod-liver oil or malt extract are indicated.

Where plethora and constipation are present, a wineglassful of Friedrichshall water before breakfast should be taken. If the liver is congested, or functionally deranged, a drachm of Carlsbad salt in a tumblerful of warm water should be sipped while dressing in the morning. Lauder Brunton has shown that the sipping of even water alone stimulates the biliary flow.[1] Other purgative salines, and liquor potassæ, are also helpful.

Alcohol should be studiously avoided during the period prior to the discharge of the core, as it increases the throbbing pain considerably. Of course, there are cases in which a little stimulant may be necessary from the great debility of the patient.

Prof. Spence[2] advises the administration of colchicum in small doses, and saline purges, followed by regulation of the bowels by rhubarb, and potash or soda bicarbonates, when hot skin, scanty urine, and constipation exist together in an attack of furunculosis. Excessive acidity or alkalinity of the urine should be counteracted by alkalies and dilute mineral acids respectively.

As to drugs having a direct influence on the process, the one

[1] *Disorders of Digestion.* [2] *Lectures on Surgery*, vol. i., third edition.

which deserves first notice is undoubtedly arsenic. This von Tröltsch[1] strongly asserts to possess specific properties in furunculosis. Erichsen[2] recommends this drug especially when boils are associated with pompholyx, or preceded by painful vesicles. It is conveniently given as Fowler's solution.

Charcoal has been recommended, probably for its absorbent and oxidizing properties, in cases of alimentary disturbances, and those associated with the production in the gut of gaseous, animal, alkaloidal, or other products of perverted digestion. Bragg's charcoal biscuits form an agreeable and "elegant" way of administering it.

Calcium sulphide is given by some surgeons in doses of one-tenth of a grain every hour till three or four doses have been given. Ringer believes that it helps the separation of the slough, and is also prophylactic in its action. It is, however, an extremely disagreeable drug to administer, and certainly gives no better results than arsenic.

Yeast is a well-known "household remedy," in doses of one tablespoonful thrice daily. It is very disappointing in the results it gives, so far as my experience of it extends.

Prof. Chiene, of Edinburgh, recommends that perchloride of mercury should be given in cases of whitlow, boils, and carbuncles, as an antifermentative. For this purpose, in several cases which I considered, from the history of alimentary symptoms, to be due to absorption from the gut of irritating products, I administered Liquor hydrargyri perchloride in 3j. doses, and it certainly proved helpful. I now prefer to give a good dose (say grs. v. or vi. to an adult) of calomel, which has the additional advantage of evacuating the contents of the intestine; while, by its partial conversion into mercuric perchloride, it produces its effects in the gut, blood, and skin as a powerful antifermentative agent, and locally as an antiphlogistic.

As to general prophylaxis, I may add that Berkeley Hill[3] states that the mineral waters of Vichy, Barèges, or Harrowgate are believed to remove the disposition to the recurrence of boils.

The *local treatment* of furunculosis is in the main merely protective, the object being to produce a state of local rest, and thus limit the effusion, and perhaps also the amount of sphacelation, by minimizing the violence of the local inflammation. In addition to this, irritation of the inflamed surface by mechanical irritants, such as woollen or other under garments, is lessened. These ends are generally aimed at by the application to each boil of galbanum and opium, lead, belladonna, or soap plasters, or the emplastrum roborans (ferri),—a small opening being generally cut in the centre of the plaster to admit of the escape of the infective pus, which otherwise spreads underneath the plaster, with a remarkably dis-

[1] *Lehrbuch der Ohrenheilkunde.* [2] *Op. cit.*
[3] *Op. cit.*

agreeable result. This treatment is practically expectant. It cannot arrest the process, and assists very slightly in its lessening or resolution; and it almost invariably proceeds to pointing and "bursting." It too often happens that at night, during sleep, the boil "breaks," with a more or less profuse discharge of pus, which bathes the neighbouring skin, and produces, by infection of the hair follicles, an abundant crop of boils round the original one. This is a very frequent experience. In every sense the merely protective mode just described is unsatisfactory, though it is stated by some surgeons to give great relief to pain, by its protecting the boil from the chafing of the under garments.

Poulticing, as generally (and far too often) carried out, is generally condemned by most careful practitioners in the treatment of boils. The warmth, moisture, and nourishment supplied by the ordinary bread, bran, or linseed poultice directly favour the growth of the specific organism of furunculosis. The moist heat promotes increased softening of the tissues and liquefaction of the inflammatory products, and hence leads, on the bursting of the boil, to a larger discharge of infective fluid. Even if such poultices be sprinkled with 1–20 carbolic lotion, they are not altogether safe, for the small amount of antiseptic fluid is soon evaporated by the increased local temperature. Corrosive sublimate lotion (1–2000) would, *à priori*, seem to be less objectionable from the non-volatility of the salt. Again, covering the surface of a linseed or bread poultice with a layer of grease, vaseline, or other oleaginous material, for the purpose of preventing infection of the neighbourhood, is of no use, although often practised; for if the boil bursts, infection will certainly occur. Poultices well saturated with port wine do not tend to decompose so readily, as the alcohol and many of the volatile ethers (œnanthic, tartaric, malic, racemic, acetic, pelargonic, and others) are antiseptic, but are volatile. Dilute acetate of lead solution is occasionally also sprinkled on poultices for its sedative effects, which are, however, slight and temporary. Starch poultices have been recommended as producing less pustulation than others. Sir James Paget recommends resin cerate, spread on a poultice, as a useful palliative. But, if poultices are to be applied, the preference should be given to one formed of four or five layers of boric lint, covered with guttapercha tissue or oiled silk, and over all plenty of cotton wool, so as to prevent too rapid radiation, and to equalize the pressure of the bandage which keeps it in position. Such a poultice is efficient from the non-volatility of the antiseptic, and from its not yielding any pabulum to germs, supposing any have escaped or survived the onslaught of the boracic acid! Hot fomentations—of flannel or spongio-piline—are objectionable if left on too long, as they so often are. The *rationale* of poulticing and fomenting is the hastening of the separation of the slough by the softening and liquefaction of the tissue surrounding it; and also the relief of pain

due to the impact of the blood current against the arteriole walls, and block of leucocytes in the capillaries,—*moderate* heat and moisture tending to dilate these vessels, and generally to lessen local action and diminish tension.

I think it was Professor Spence who first recommended a very simple procedure, which never fails to yield great comfort and benefit in cases in which there is much tensile pain in the boil. This is the application of a small sponge wrung out of water as hot as it can be borne, and its frequent re-application every four or five minutes, so as to keep up the temperature of the sponge to a point sufficient to secure tonic contraction of the vessel-walls. In many cases even this is not necessary if the procedure detailed further on be appropriately and timeously employed, but it does away with the necessity of a most agonizing method of treatment, viz., incision,—too often advocated, and more often practised.

Incision of a boil undoubtedly promotes increased loss of tissue, and greatly contributes to the formation of much more disfiguring scars than those which occur if the boil is left to itself, and allowed to point and discharge. Early incisions are especially to be deprecated, for there is no slough formed then whose discharge might be facilitated by an incision.

The frequent application of a small piece of lint, the size of the boil, and soaked in cold water, has in many cases a soothing effect, but does not tend to shorten the process materially. Of course, it is dangerous in any but the early stages, from the risk of infection of the neighbouring hair-follicles if left on too long.

Equal parts of extract of belladonna and glycerine painted on the boils is a very favourite palliative, and very widely resorted to. I confess I have never seen any good results from it, save a slight diminution of local pain. I have also tried, with private patients, the application of a four per cent. cocaine solution, and it certainly gives a little relief, though the pain is often too deep-seated in the cellular tissue to admit of palliation thereby.

After the prevention of the dyscrasic condition which produces boils by appropriate constitutional and hygienic treatment and a proper dietary have failed, the abortion of the local process is the ideal to be aimed at. Sir Peter Eade, of Norwich, has attempted this by the application of a drop of pure carbolic acid to a commencing boil, especially if in the vesicular stage. I remember Dr Allan Jamieson employed a similar mode in dealing with molluscum contagiosum in the Skin Department of the Edinburgh Royal Infirmary. The analogy between furunculosis and molluscum contagiosum lies in the facts that both are sebaceous gland affections (with, in the latter case, a specific degeneration of the rete Malpighii), and depend, in great part, on germ-infection for their *raison d'être*. I have followed this mode of treatment for more than three years, and with a very fair proportion of successful results. It is a thoroughly rational mode of treatment, aiming as it

does at the destruction of the specific furuncular organisms in and about the hair follicle and its glands by the caustic and antiseptic action of the diffusible carbolic acid. It also, after a brief period of depression, stimulates the cells of the part (which have not been killed or disabled by it) to a mild and beneficial local action. This enables them to withstand, in many cases, the causes which are tending to produce a localized phlegmonous inflammation. But, in many other cases, it utterly fails, as I have often regretfully noticed—perhaps because applied too late. As to the best mode of applying it:—First epilate the hair projecting from the papule, and which serves to mark the affected follicle; then apply with a glass rod one or more drops of the fluid obtained from the pure crystals by liquefying them at a great heat. There is a slight smarting for a minute or so, and the part is faintly whitened by the caustic action; then follows the analgetic effect of the carbolic acid, and there is no more bother with that site, unless, of course, it has failed. Nothing like ulceration—nor, indeed, any bad result—ever follows this method.[1] Other concentrated antiseptics might, no doubt, also usefully be applied, *e.g.*, a saturated solution of corrosive sublimate, or thymol, or even eucalyptus oil.

Touching with nitrate of silver stick, or painting with an alcoholic solution of this salt, has been recommended. I have never tried it, as I consider the salt is too weak a germicide, and too strong an irritant, to prove useful. The carbolic acid analgesia and its germicidal and caustic effects are powerful reasons for preferring it. Another seemingly good application is that which Dr Veiel[2] uses. It is composed of equal parts of oxide of zinc and vaseline, to which four per cent. of boracic acid is added. This is spread on lint and applied thrice daily. It is, of course, astringent and antiseptic, and should be useful.

After the separation of the slough—especially if large—dressing with Peruvian balsam, and protection with compresses, is recommended.[3] I prefer Ungt. Galbanæ Co. for its local mild stimulant properties; or Tinct. Benzoinæ Co.; or terebene and olive oil (1–3).

I have now reviewed the modes of treatment in vogue, so far as my recollection and experience of them will allow, and now come to consider a strikingly simple as well as, I think, a rational mode of treatment, which has yielded uniformly successful results in a considerable number of cases. It is a combination of local antisepsis and massage, combined with the ordinary constitutional treatment already adverted to. In the early stage, before actual

[1] I should add that the views here stated are solely my own. I have not seen Eade's remarks on this subject. I only know that he had recommended pure carbolic acid in the vesicular stage of furunculosis.

[2] *Monatsh. f. prakt. Dermat.*, vol. xi. No. 8, 1890.

[3] Erichsen, *op. cit.*

sphacelation has occurred, and when we have to deal with a condition of effusion in the vicinity of the hair follicle, which has been excited by the action of pathogenic germs, it will be obvious that we should be able to arrest and abort the whole process by destroying the germs by a powerful diffusible antiseptic, and then driving the effusion back into the lymphatics of the part by massage; while, at the same time, we raise the standard of tissue vitality, by measures calculated to improve nutrition and tissue metabolism, to a state in which it can successfully combat the morbid tendency. I therefore proceeded to apply these principles to several incipient boils which had resisted the application of pure carbolic acid. The first case I employed it in was that of a very senior medical officer, under whom I was at the time serving. The result was most gratifying, and several incipient boils were aborted entirely. This officer cordially agreed with me in the efficacy of the procedure.

The method is as follows:—The boil and skin adjacent to it is first soaked with glycerinum acidi carbolici (P.B. strength, 1 to 4; or, if the patient's skin is known to be tender or irritable, 1–10 or 1–12 may be substituted); then it is *gently* worked into the skin over and for an inch or so round the papule or commencing boil, beginning in a small circle round the hair follicle, and working out in gradually diverging circles. The pulp of the finger is admirably adapted for this purpose, being soft and sensitive. The pressure and manipulation are quite bearable, and, after a minute or so, the friction soothes the local pain, probably by relieving the pressure on the nerve filaments of the effused serum and "coagulable lymph," which are squeezed out of the part and driven on into the lymphatics for removal. At the same time, the glycerine softens the tense skin, and perhaps also asserts its hygroscopic properties, thus further relieving tension; and, no doubt, the carbolic acid also exerts mildly its analgetic properties. The massage is continued for ten minutes; then a layer of lint, thoroughly soaked in the carbolized glycerine, is laid on the part, and fixed there, and in two or three hours the whole procedure is repeated. This is continued at intervals till the whole process is resolved. In a very few hours in many cases, and in others on the following day, all signs of the incipient boil's site have gone, and the chance of recurrence in adjacent follicles is prevented by their antiseptic condition, and the direct stimulation of the cutaneous circulation round about them by the massage. I have not as yet observed any symptoms of carbolic acid poisoning, although I have thus treated cases of multiple boils distributed very widely over the body. This treatment is emphatically designed for the early stages: *when a slough has formed, it can be of little or no use.*

But it is possible to recognise every papule likely to become a boil by the presence of more or less effusion and hardness round it, which is not present to anything like the same degree in other

papular skin affections. When such a condition has been recognised, the abortion of the boil should be effected by the means above stated.

Another point aimed at is the prevention of local recurrence round about the diseased area. Nor must it be forgotten that the constitutional treatment of the furuncular dyscrasia is equally important. Should the condition have gone on to sphacelation, and pus is being discharged, the carbolic glycerine or eucalyptus oil or other antiseptic is essential for successful treatment, applied on lint and conjoined with the frequent cleansing of the part. Eucalyptus oil is a reliable and diffusible antiseptic, which exerts a bland and unirritating local action. In addition, there is no doubt that gentle massage, as above described, is decidedly *helpful* at this stage.

Throbbing or cutting pain can almost invariably be relieved by the application of a small sponge to the part, as already described: the hot water out of which the sponge is wrung is already rendered aseptic by boiling. I never have to resort now to an incision in my cases of furunculosis of the trunk or limbs.

Aural furunculosis, of course, defies the method of treatment I have advocated. My almost invariable procedure here is one I have just stated I have abandoned elsewhere, viz., incision, followed by hot boric douches. The tension in these cases is generally so great, and the relief given by incision so instantaneous, that it seems the best mode of treatment at present. A fine von Graefe knife is the most convenient instrument for the purpose. Antisepsis by such means as a concentrated alcoholic solution of boracic acid has been practised,[1] and palliation with Gruber's gelatine bougies medicated with extract of opium;[2] or hot-water instillations, or a cotton-wool plug soaked in glycerine[3] are also practised, but are all inferior to incision.

Syphilitic furunculi are to be treated by constitutional remedies, such as mercury or potassium iodide; and, locally, by mild mercurial ointments, such as those of the yellow oxide, or the red iodide (diluted, in the latter case, with vaseline or lanoline).

The method I have advocated in this paper is readily and universally applicable in all classes of cases—in hospitals, by nurses or other sick attendants; and in private practice, by the patient's relatives or friends. It no doubt involves a great deal more patience, exertion, and fatigue on the part of the patients' sick attendants, but surely the first thing to consider is the relief and speedy termination of an extremely painful and reducing malady. And to this, the first and most obvious advantage, must be added the absence of the disfiguring cicatrices with their encircling purple pigment. These blemishes last for a very considerable time, often for two years; and of course the scars them-

[1] M'Bride's *Guide to the Study of Ear Disease.* [2] *Ibidem.*
[3] *Diseases of the Ear*, by Dr St John Roosa.

selves never disappear, but remain as pearly white puckered marks, which do not add to the personal attractions of the individual affected.

I possess notes of my early cases, but all that is of interest is already embodied in the foregoing remarks. The last case I treated may be, however, well cited as a type of many others. The patient was an officer's wife whom I had eight or nine months before treated for a diffuse crop of boils, which had spread all over her back, face, neck, and arms from a single boil on the elbow. I treated her constitutionally, and with plasters and extract bellad. and glycerine paint. She came to me a week or two ago with a single boil on the shoulder which was fairly well advanced. The method was carried out for a day and a half, and every trace of the boil speedily disappeared, and she had no others, which she would inevitably have had, I am convinced, if I had treated her by the methods I used before my employment of the massage with carbolized glycerine.

In conclusion, I can only reiterate my firm conviction of the usefulness and efficacy of the method now detailed. My experience of it extends only for six or seven months back, during which the results it has given, however, have been most encouraging.

Meeting IX.—June 3, 1891.

Dr Smart *in the Chair.*

I. Exhibition of Patients.

1. *Dr Allan Jamieson* exhibited a favous family. He had attached this name because all the children, five in number, had suffered from favus. The eldest was twenty, and had had the disease sixteen years when he came under treatment; the youngest was seven, and had been affected two years. The cure had occupied from six to eighteen months, and six months had been allowed to elapse ere he had presented them to the Society, so as to ensure its permanency and completeness. No diseased hairs had been found for that space of time. In treating them—first, the hair had been kept very short, and the heads had been washed daily with a superfatted potash soap; second, all diseased hairs had been epilated over and over again; third, a paste consisting of one drachm each of resorcin and oxide of zinc, and two of lanoline, vaseline, and starch, had been thinly applied after the head had become dry. He would particularly direct attention to the pliancy and mobility of the still bare parts, and to the fact that these were being to some extent covered anew with hair.

2. *Dr Smart* showed a woman trephined for basal hæmorrhage

when nearly dead. The patient, a woman in her 46th year, was brought by the police to Ward VI. on June 22nd, 1890. She was conscious, and answered questions fairly well. There was a bruise on the right side of the head, near the parietal eminence, where she had received a blow. The face was drawn to the right, but without ptosis. There was no ocular deviation. The pupils were nearly equal, and reacted fairly to light; but it was evident that there was only half-field vision when tested by the rough method of using the fingers. In other words, there was left hemianopsia. She could not raise the left arm, nor grasp with the left hand, the left forearm remaining flexed and the fingers curving upon the palm. The thigh and leg movements were defective in a less degree, and the sensibility of both arm and leg, as also of the whole of the left side, was irregular and deficient. Speech was somewhat slurring, but not aphasic. Some hours after admission coma set in, and some hours later Cheyne-Stokes breathing supervened, and soon became so grave as to threaten life. At his request Prof. Chiene trephined, first at the place where the blow had been received, and a second time further forward and a little lower. There was free hæmorrhage from the membranes, but no discoverable cause was found within reach of the director or finger to account for the symptoms. This result corroborated his diagnosis of a right basal lesion as determined by the existence of a left hemianopsia. The breathing, characterized before the operation by lengthened intermissions, and well-nigh suspended, had become almost normal before the operation was completed, and thereafter continued to improve. This effect was especially noticeable after incising the dura, thereby giving relief to the brain pressure. The patient, a few hours after the operation, had regained consciousness, and continued to improve as regards her intelligence, until the month of August, when she left the Hospital, in complete possession of her faculties, the hemiplegic disability above referred to continuing, with the addition that the left intercostal muscles were now observed to be involved in the paralysis affecting the left side. In May of the present year she came to the Hospital to show herself, and was re-admitted to Ward VI. to undergo electro-massage treatment for the hemiplegia, which remains in much the same condition as when she left. In other respects she enjoys excellent health, and her intelligence is unimpaired. So far as known to him, this was the first example of trephining for a basal lesion, the case being diagnosed from the beginning as an apoplexy at the base. The success attending it would doubtless encourage to the performance of that operation in similar cases, and, speaking generally, widen the basis whereby that operation was rendered justifiable. Since the patient's return to Hospital, Dr George Mackay, Assistant Ophthalmic Surgeon, Royal Infirmary, had made a most careful perimetric ocular examination, with the result of showing that the condition of left hemianopsia, above referred to, originally diagnosed, still continues.

II. Original Communications.

1. ON RELAPSE OR RECRUDESCENCE IN SCARLET FEVER: TWO CASES, WITH A NOTE ON THE LITERATURE OF THE SUBJECT.

By George P. Boddie, M.B.

Various eruptions occasionally appear during the course of an attack of scarlet fever—*e.g.*, roseola, erythema, urticaria. These may be due to dietetic errors; to external applications, such as turpentine, camphor, menthol, carbolic acid, and irritating soaps; or to other causes. Some of these eruptions may even be followed by partial desquamation, but a true relapse or recrudescence in scarlet fever, as opposed to a recurrence, is comparatively rare. When such does occur, important points of diagnosis, prophylaxis, and treatment are necessarily raised.

The following two cases seem to be of this nature, and are of sufficient interest and importance to bring under the notice of the Society:—

Case I.—A boy of 14 years of age was, on the 5th of May 1890, seized with shivering, nausea, and headache. On the evening of the following day, when the patient was first seen (I was asked to see him in the absence of Dr Ronaldson, and I am indebted to his kindness and courtesy, not only for allowing me to see the case from time to time, but for permitting me to quote it), his temperature was 102°·4, pulse 118, respirations 26, and he was complaining of headache and sore throat. There was a bright red eruption of very minute points on the sides of the neck, chest, left axilla, down the left side, the left shoulder and arm, and on the right leg. The tongue was coated, and the papillæ were prominent; the tonsils were inflamed, swollen, and patchy; the hard palate, fauces, and pharynx were deeply injected. The cervical glands were swollen and tender. The temperature subsided gradually, and was normal on the sixth day, as you will see on the Chart. On the eleventh day the hands and feet had a shreddy look. On the thirteenth day the cuticle began to separate as a fine scurf, with a few flakes, on the sides of the neck, arms, fingers, and toes. This went on for some twelve days; the desquamation was only partial, but was not confined to the surfaces affected by the eruption. On the fifteenth, sixteenth, and seventeenth days there was slight albuminuria.

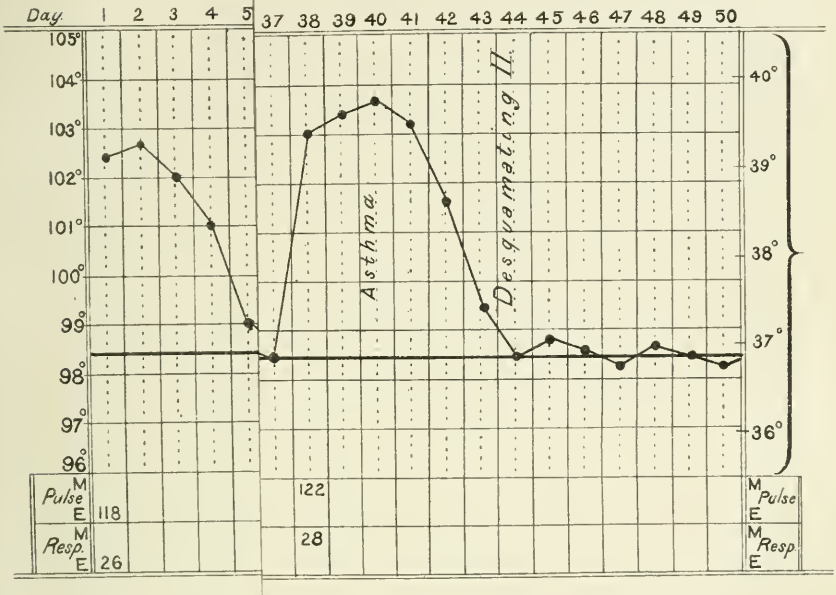
Two children living in the same house passed through undoubted attacks of scarlet fever. A girl, who slept a night or two in the house, was taken ill 48 hours after she left, and was removed to the City Hospital suffering from well-marked scarlatina. Other four persons living in the house suffered from sore throats accompanied by painful cervical glands. The surroundings thus seemed

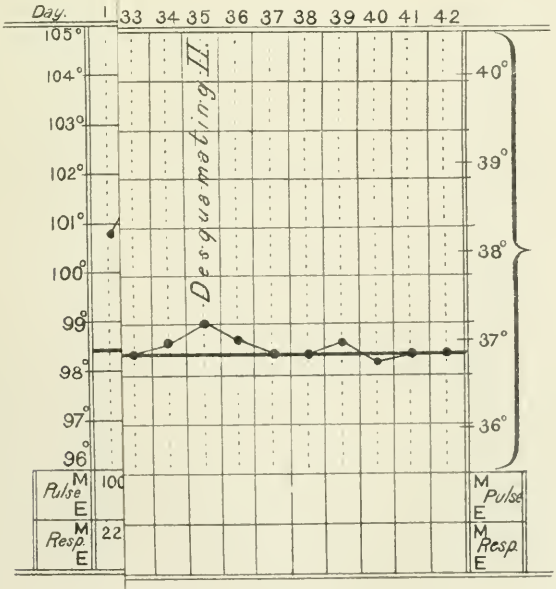
to leave no possible doubt with regard to the diagnosis. Evidently the boy in question had a distinct attack of scarlet fever. Dr Heron Watson, who was attending a surgical case in the house, was good enough to see the boy twice with me, and he confirmed the diagnosis.

Now for the further history. On the 11th of June, that is on the thirty-seventh day, the patient was apparently so completely well that he was permitted to go to the hall of the Edinburgh Exhibition to see the freedom of the city conferred on Mr Stanley. The day, as some may remember, was cold, wet, and raw in the extreme, and the wind east. On the evening of that day he complained of slight shivering and headache. His temperature was rising. Next day the temperature was 103°, the tongue red, the throat again injected, the cervical glands enlarged and tender, though to a less extent than during the former attack. The trunk and extremities showed an irregularly distributed papular red rash, which began to fade on the morning of the third day. Desquamation began on the seventh day, and was more extensive than in the former attack. At the end of five weeks, 72 days from the first illness, the boy was perfectly well.

A point of some interest in connexion with this case may be noted. The boy had for several years been subject to frequently recurring attacks of bronchial asthma. During the initial attack of scarlet fever he was entirely free from asthma, but on the third night of the relapse he had a sharp seizure. Curiously enough he has never had an attack since; and whereas formerly the slightest deviation from a rigid diet brought on asthma, now he is able to eat almost anything with impunity.

Case II.—I speak of the second case in terms of much greater reserve than the first, because, as you will at once see, it presents some points difficult of explanation. It is of interest not merely because it seems to illustrate the occurrence of a relapse of the scarlet fever on the twenty-eighth day, but because there also apparently intervened a concurrent attack of chicken-pox between the initial scarlet fever and the relapse. I give you briefly the main points. A boy of 9 years of age took ill on March 24, 1891,—rigor, sickness, headache, sore throat, swollen and tender cervical glands; temperature when first seen 100°·8, rising later to 102°·6; the body almost entirely covered with a perfectly characteristic scarlatinal eruption, which faded on the third and fourth days. Some school companions were known to have scarlet fever, and two others chicken-pox. A lady whose two children were ill of chicken-pox called at the house on the second day of his illness, but did not see the boy. Desquamation began on the ninth day. During the night of the tenth day he was taken ill, had a sharp rigor, complained of severe headache, vomited, felt extremely weak, and his skin was reported to have been very hot. Next morning his temperature


Day. 1 2 3 4 5 37 38 39 40 41 42 43 44 45 46 47 48 49 50
105° 104° 103° 102° 101° 100° 99° 98° 97° 96°
40° 39° 38° 37° 36°
Asthma.
Desquamating II
M Pulse E 118
M Resp. E 26
122
28
M Pulse E
M Resp. E


Day. 1 33 34 35 36 37 38 39 40 41 42
105° 104° 103° 102° 101° 100° 99° 98° 97° 96°
40° 39° 38° 37° 36°
Desquamating II
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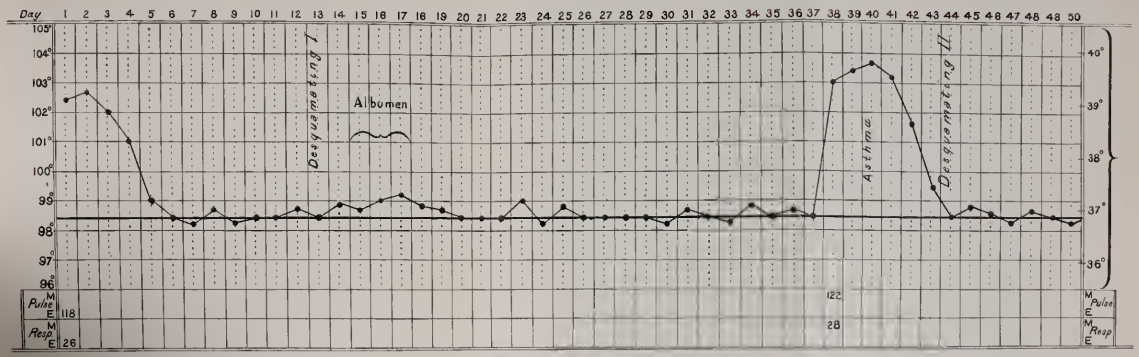
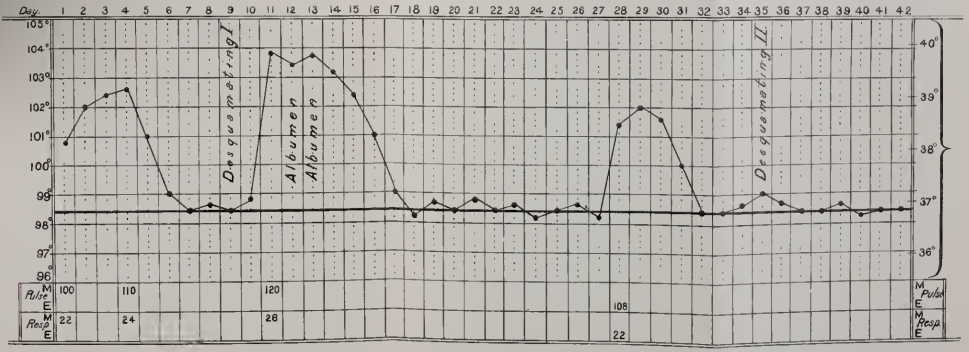
M Pulse E
M Resp. E

Relapsing Scarlet Fever: Geo P Boddie

Case I



Case II



was 103°·8, pulse 120, respirations 26, and he looked very ill. On the face, chest, and wrists a number of vesicles of various sizes, from the traditional millet seed upwards, were seen. During the next four days successive crops of vesicles of larger size appeared. They were distributed over the whole body, on the eyelids, on the conjunctivæ, and on the hard palate. Many of the larger vesicles contained pus. Slight albuminuria appeared on the twelfth and thirteenth days. The whole condition was so marked as to be suggestive even of small-pox. The boy was practically well on the twenty-first day, although the formation and separation of scabs (as well as desquamation) was going on. He remained well till the twenty-seventh day, on the evening of which he was feeling out of sorts. On the twenty-eighth day the temperature rose to 101°·4; there was again slight nausea, loss of appetite, some headache, sore throat, and the appearance of an irregularly distributed red papular eruption. The temperature was normal on the thirty-second day, and practically remained so, as you will see from the Chart. A second desquamation began on the thirty-fifth day; it was partial, and was apparently quite over by the forty-second day.

You will have gathered from what I have said that in the first case the relapse was much more severe than the initial attack; while in the second case the relapse (if so it were) was much milder and shorter. This irregularity seems to be the general experience in such cases.

Thus Rilliet and Barthez write, "Aucune donnée ne peut faire prévoir la gravité de la récidive; elle peut être plus légère ou plus grave que la première, voire même se terminer par la mort" (*Traité Clinique et Pratique Maladies des Enfants*, iii., 154).

In connexion with these cases, it may be of some interest to the members of the Society if I run rapidly over the more important literature of the subject.

One of the first, if not the very first, fully reported case was that of a girl of 3½ years, under the charge of West in the London Hospital for Sick Children, seen by Jenner, and reported by Hillier. The child had measles on June 16, 1854, and a second eruption with all the characters of scarlet fever on the 23rd and 24th of the same month; left the hospital tolerably well on July 21. A week later, *i.e.*, thirty-six days after the first scarlatinal eruption, she was brought back with a papular red rash, which Jenner considered to be that of scarlet fever. Desquamation was taking place. On the forty-fourth day she died from pneumonia. Hillier refers to other two cases,—one a medical student who had had scarlatina three times, and a distinct relapse a week after the third attack; the other a young woman who had a relapse five weeks after the initial attack.

Sir William Jenner, in a clinical lecture on scarlet fever, given at University College Hospital, and published in the *Lancet* of 1870, says:—"Here, again, is the note of another case, ... but

in one particular a rare case. I have seen several such, and believe they are more common than is supposed. It is a case of relapse; the patient had scarlet fever twice in a short time" (the relapse occurred on the twenty-second day). "As a rule," he adds, "the second attack runs a short course, but I have seen a patient die in the relapse."

Further references in British medical literature are of the most meagre description. Few of the larger text-books even refer to the subject.

Hilton-Fagge asserts that "relapses are seldom or never seen" (*Principles and Practice of Medicine*, i. 216).

W. Squire, in Quain's *Dictionary*, gives the brief note:—"A recrudescence or relapse is possible as late as the fourth week; this, if not a reinfection, prolongs the duration of the infectious period."

Goodhart, in his *Student's Guide to the Diseases of Children*, says, "Relapses are not very rare. Hillier mentions the case of a student who had had three attacks of scarlatina, and a week after his third attack he had a distinct relapse. Thomas describes pseudo-relapses in which a roseolous eruption breaks out after the fever has run its course. They generally terminate favourably." But Thomas also describes true relapses, as we shall show later.

E. O. Hopwood, of the London Fever Hospital, writes, "A relapse, or true second attack of scarlet fever, occurs in about one-half per cent. of cases at any time from the tenth day of the first attack, and is followed by a second desquamation. It may be of any grade of severity" (*Dict. Pract. Med.*, Ed. J. K. Fowler).

Ashby and Wright, in their *Handbook on the Diseases of Children*, p. 213, give the sceptical sentence—"Reinfection or relapses are said to take place in some instances; in cases coming under our notice (*i.e.*, in the Manchester General Hospital for Sick Children), which have had an attack of genuine scarlet fever after admission to our fever ward, there was considerable doubt as to the correctness of the original diagnosis."

Dr David Page, in his *Facts about Fevers*, gives a case of relapse after the fortieth day.

Dr Barrs, of the Leeds Fever Hospital, gives, in 1883, full notes of a case of "complete relapse" on the thirty-first day.

Several other cases are reported in the *British Medical Journal*, the *Lancet*, the *Medical Times and Gazette*, the *London Medical Record*, and other journals, during the last twelve or fourteen years.

The subject has received wider recognition in American medical literature.

In the *Cyclopædia of the Diseases of Children*, edited by Keating, the following passage occurs:—"Second attacks must be distinguished from the cases of relapse which follow immediately in the wake of the first attack, and also from the cases of pseudo-relapse,

which are characterized by the recurrence of the exanthem during the second or third week of the disease."

The writer of the article on scarlet fever in Pepper's *System of Practical Medicine* says, "Sometimes a second attack occurs so soon after the first that it has been described as a relapse." He quotes two cases, one of the two being a case of Hillier's already alluded to.

The references in American journals are too numerous for quotation. A note of the more recent of these will be found in the *Annual of the Universal Medical Sciences* for 1889.

We come now to what may be fairly described as the *locus classicus*, viz., the work of Thomas, Körner, and Trojanowsky.

We quote first from Thomas, who has recorded several cases. "As analogous to the relapses of typhoid fever, those cases in which the second attack sets in before the patient has entirely recovered from the first may be designated as true relapses. Few of the reports inform us regarding the number of days which intervened between the cessation of febrile action and the symptoms of the fresh attack. I do not think, however, that we shall err if we include in this category all those cases of a second attack which are reported as having occurred immediately after the first one, and those also in which it is stated that the fresh affection began not later than four or five weeks after the first. Such true cases of relapse have been described by Bartels, Barthez, and Rilliet, Faye, Gaupp, Jenner, Hillier, Kjellberg, Lefèvre, Müller, Marshall Hall, Peacock, Richardson, Röbbelen, Schwarz, Smith, Solbrig, Steinbeg, Steinmetz, Steinthal, Stiebel, Trojanowsky, Wood, and an unknown author, according to Thomson."

"In the majority of cases the first and second attacks were developed with equal completeness; in some cases the second attack was more rudimentary; in a very few cases the first attack was not entirely characteristic. At one time the report states that the first, at another that the second attack was the severer of the two; in several cases the greater intensity or mildness of the subsequent attack seems to have been determined by the contrary behaviour of the antecedent attack, so that both seem to have completed each other . . . . . With the exception of several cases reported by Jenner, Peacock, and Smith, who observed fatal results in children, the relapses of scarlatina always ended in recovery. When the second attack occurs later, it seems that recoveries are still more frequent; at least, I can find but one report of a fatal result, that of West and Hillier, in which the second attack began thirty-six days after the commencement of the first."

So much for true relapse (*ein wahres recidiv*). But Thomas, and also Körner, have described what they call pseudo-relapse (*ein pseudo-recidiv*). I again quote Thomas: "Sometimes after an attack of scarlet fever, which runs a typhoid course without special local symptoms, or is attended by various local diseases, or perhaps is

uninterrupted by any complications, a fresh eruption unexpectedly breaks out over the whole body. In the cases which have come under my own observation, it is never exactly similar to the normal first scarlatinal rash, and yet the resemblance is so strong, and the difference from all other exanthemata so marked, that I have no hesitation in regarding it as an irregular second scarlatinal eruption, and shall therefore distinguish it from the true relapse, which occurs after the normal course of the disease, by calling it a pseudo-relapse. The cause of this anomalous eruption may probably be found in a renewed determination to the skin occurring at an unusual time. Nephritis appears to have no influence in producing the pseudo-relapse, at least this is the experience of myself and others . . . . . During the continuance of the second exanthem, the course of the body temperature is by no means regular and characteristic. This fact may perhaps by itself enable us to distinguish the nature of the new process. I have noted particularly that the maximum of the pseudo-relapse never coincides with the maximum of the temperature." — Ziemssen's *Cyclopædia*, vol. ii., *in loc.*

Trojanowsky (*Jahrbuch für Kinderheilkunde*, 1873) states that among 300 cases of scarlet fever (260 children and 40 adults) he has seen 18 relapses (15 children and 3 adults). That is, you observe, 6 per cent. He has published full notes of ten of these cases. Trojanowsky has further described a recurrent form of scarlatina which is peculiar. "From seven to seventeen days after the first eruption of scarlatina, a second eruption similar to the first, or identical with it, makes its appearance, so that both attacks combined seem to form one scarlatinous affection. Both attacks were accompanied by an unusually high fever, similar to that of relapsing fever, and separated from each other, as in the latter disease, by a complete remission, during which all morbid signs disappeared, again to recur with the commencement of the second attack; in addition to the ordinary symptoms of scarlatina, the spleen, as in relapsing fever, increased rapidly in size from the commencement of the attack, the white blood cells became abnormally numerous, and there was extraordinary prostration together with muscular pains. Now, as most of the patients affected in this manner lived in low and marshy localities, in which relapsing fever occurred almost every year, and also to a certain extent during epidemics of scarlatina, we may freely assume that these cases were the expression of a peculiar combination of relapsing fever and scarlatina."

Körner, of Leipzig, in 1876, published an elaborate paper in the "*Jahrbuch für Kinderheilkunde*," in which, following Thomas, he distinguishes pseudo-relapse (*ein pseudo-recidiv*), true relapse (*ein wahres recidiv*), and true second attack (*eine zweitmalige Erkrankung.*) "*A true relapse* in scarlet fever," he says, "is quite analogous to the relapse in typhoid (abdominal typhus); the first

febrile process is completely gone, usually desquamation has appeared, sometimes even terminated, when there sets in a renewed manifestation of the disease. There appears for the second time a characteristic scarlatinal exanthem, all the symptoms of the illness begin anew, sometimes worse than in the first illness, often the new exanthem completes the former." Körner supplements his paper by giving notes of thirty-eight cases of true relapse, as reported by the following writers,—Bartels, Barthez, Eisenmann, Gaupp, Marshall Hall, Hillier, Jenner, Krauss, Kjellberg, Lewin, Landeutte, Lefèvre, Müller, Rilliet, Richardson, Röbbelen, Solbrig, Steinthal, Steimmig, Schwarz, Shingleton, Steinbeck, Stiebel, Steinmetz, Thomas, Trojanowsky, West, Wetzler, and Wood. He also gives notes of five cases of pseudo-relapse, and a long list of cases of true second attack.

Gumprecht, in the course of his experience at the Friedrichshain Hospital, made careful observation of 228 cases of scarlet fever, and in this number met with 13 "pure cases of relapse," *i.e.*, 5·7 per cent.

Fürbringer, who cites Gumprecht's calculation in the *Real-Encyclopädie der Gesammten Heilkunde*, 1889, says that, in the course of his 14 years' experience in Baden and Thuringia, he only met with occasional cases, and it always appeared to him that the relapse was "a fever following the true initial scarlatina, independent of it, caused by no apparent complication, of very variable type, and lasting for days or weeks."

Reimer, of St Petersburg, in the *Jahrbuch f. Kinderheil.* for 1876, published a detailed report of a case in a boy of 7. The relapse occurred on the eighth, and death on the tenth day. The attack was complicated by pneumonia and hæmaturia. The second exanthem and the hæmaturia appeared coincidently.

Huettenbrenner, of Vienna, reports that he has seen several cases.

I must omit several other references. Henoch's statement, however, is so valuable that I make no apology for quoting it in full.

"Although not so commonly as in typhoid, still relapses do occur in scarlet fever. After the patient has been quite free from fever for several days, or even weeks, and desquamation has begun in the normal way, the rash suddenly sets in afresh, either over the whole body or only on some parts of it, the temperature again runs up, and the disease goes through its course a second time. In such relapses the symptoms may be more severe than they were in the first attack. The reddening of the skin during desquamation is very interesting, and has a quite peculiar appearance. Practitioners have given more and more attention to these relapses since the publication of the observations of Trojanowsky, Thomas, and Körner; and I have seen at least a dozen cases. I shall only mention the following." Here follow notes of eight cases. He comments on these so:—"In these cases we cannot speak of a fresh infection any more than we can do so in the relapses of typhoid

fever; and I can only account for them by supposing that the scarlatinal virus had not been completely eliminated by the first attack, and that therefore a relapse was inevitable. If stricter attention were given to the subject, it would probably be found that relapses are much commoner than is generally supposed. But you must not expect to find in every instance such a typical series of symptoms as in our first case. We are more likely to find single symptoms, fever or rash, appearing so transiently that they are apt to be overlooked, especially in practice among the poor. On the other hand, we must take care not to mistake simple erythema or urticaria (which I have often seen following scarlet fever) for a relapse; because with the latter there is invariably renewed desquamation. In any case we must not regard the relapse as at all less important than the first attack. Several cases, including some in my own practice, seem to show that a relapse may end fatally from pneumonia, or with malignant symptoms, even when the primary attack has had a perfectly normal course" (*Lectures on Children's Diseases*, transl. J. Thomson, ii. 232, *et seq.*)

A word or two on French literature and I have done. The condition has been fully realized, and many cases have been reported by Jaccoud, Lefèvre, Rilliet, E. Barthez, M. Laugier, A. Sanné, and by several other writers.

Thus Jaccoud, in his *Traité de Pathologie Interne*, 1883, iii. 558, makes the following comments:—"Scarlatina is capable of recrudescence or relapse like typhoid fever. This relapse, which must not be confounded with a recurrence, takes place during the period of desquamation, or immediately after: the attack in question is not a second infection, but a redoubled manifestation of one and the same single poisoning. This relapsing scarlet fever, quite analogous to relapsing measles, has been positively demonstrated by the works of Trojanowsky, of Thomas, and of Körner."

M. Maurice Laugier reports (*Gazette Hebdomadaire*, 1871) a case of a patient who, on the fifteenth day of a comparative mild scarlatina, had a relapse which assumed a malignant type, with hæmorrhages, and was rapidly fatal.

In their *Traité des Maladies des Enfants*, 1891, iii. 153, Rilliet and Barthez write:—"The older authors denied the possibility of relapse, but we possess well authenticated cases. Wendt, Richardson, Noirot, Meissner, Wood, Heyfelder, Unterberger, May, and other authors have, as well as we, produced examples."

Dr P. A. Young said he had much pleasure in expressing his obligation to Dr Boddie for bringing this interesting subject before the Society. He had been in practice for twenty-four years, and had during that time seen several hundred cases of scarlatina, but it was only during this year that he had seen a case of relapse in this disease. During this year he had seen two cases, and, as they occurred in his own children, he was particularly interested in them.

The children, aged respectively ten and eight, developed scarlatina at the beginning of the year. The older girl had a well-marked bright rash; temperature 103°; characteristic tongue, and sore throat. In three days she had a normal temperature, and went on well, but did not desquamate freely. At the beginning of the fourth week the temperature went up to 100°, and she had a well-marked scarlatinal rash, but not so bright as on the first occasion; the temperature fell in two or three days, but the rash remained out a week. When it disappeared desquamation was much more copious than on the first occasion. The child made a good recovery. The younger girl had a temperature of 102° at the outset of the disease, and sore throat, but the rash was not well marked, and did not remain out more than twenty-four hours. She went on well, but the peeling was not well marked, although decided. At the beginning of the fourth week she also had a rise of temperature to 100°, and a rash more patchy than in the case of her older sister, and more dusky in colour. This made him at first think that both the second attacks were cases of rötheln—the former of a scarlatinal, the latter of a morbillar type. He now believed, however, that they were true relapses of scarlatina. The second girl desquamated abundantly after the second attack. He noticed in both cases after the relapse that the pulse for a week was above 100 after the rash had quite disappeared, and the temperature was normal. The children were treated in the City Hospital.

Dr James Ritchie thought that relapses should be distinguished from second attacks of scarlet fever. He believed that relapses are not uncommon during the second and third weeks, but that new attacks are very uncommon until after a very considerable period of time has elapsed. They are not unknown, and he detailed a case which came under his observation. A child, aged 12, who had passed through a very sharp attack of scarlet fever, was at the end of six weeks bathed, and within half an hour thereafter removed from hospital. The following day she had a temperature of 102°·5, severe scarlatinal throat, and copious rash. The desquamation of the first illness was not completed. She passed through a second sharp attack, and again desquamated. After an attack, protection is afforded to the majority of individuals for life, but to many the protection is only for a limited period. Whatever lowers the vitality increases the risk of a fresh infection, and in the case he had reported he believed that the warm bath and the subsequent exposure had diminished the child's power of resistance, and had rendered the fresh infection possible.

Dr James said that Dr Boddie had done well in bringing such an important subject before the Society, and that he deserved thanks for the thorough way in which he had studied the literature of the subject. Relapses in scarlet fever were, he believed, more frequent than was ordinarily supposed, and the fact that the

disease manifested itself so differently in different individuals was of importance in explaining why this should be. The disease is, of course, due to a virus entering the body and nourishing and reproducing itself there. Ordinarily, as the result probably of the introduction of some substance of the nature of ptomaines, the power of living of these lower organisms ceases after a certain definite period, and they are then eliminated. In this way the individual is protected, and it is evident that if in such diseases a protective change of this nature did not occur, the fever would go on interminably. In relapses, however, we must suppose that this protection has not been quite complete, so that before these lower organisms are eliminated they are able again to act on the individual and produce fever. In such a case the relapse occurs by auto-infection, just as it often does in typhoid fever as long as sloughs remain in the bowel. But it is possible also that a relapse may occur from reinfection. For example, a patient in a scarlet fever ward may have recovered from an attack, but yet the protection has not been complete, so that although he may have eliminated all the virus of the original fever, he is not proof against the virus of a fresh case entering the ward, and so a relapse occurs. This, he thought, applied to the case which Dr James Ritchie had just mentioned. The most marked case of relapse in scarlatina which he had seen was many years ago in Liverpool. A child was brought into hospital for croup, so ill that shortly after admission tracheotomy was performed. Some three days afterwards it was found to be affected with scarlet fever, and careful inquiry elicited that about four weeks previously it had passed through a similar attack.

Dr W. A. Jamieson.—While it was admitted that relapses might occur in the course of any of the zymotic diseases or the exanthemata, one must carefully distinguish between a relapse and a reinfection. A relapse was a relighting up of the complaint at some period within its cycle; a reinfection was a repetition of the morbid phenomena when the course of the disease had been run, or at any later period. As regarded relapses, there were differences in individual ailments; thus a relapse in enteric fever could not be well placed in the same category as one in scarlet fever. In enteric fever a relapse was concurrent with some renewed disturbance in the intestinal glands, and, as a rule, occurred early in convalescence. In scarlet fever there might be a recurrence of some of the symptoms ere the disease had proceeded very far, this he would consider as a relapse; but should all the phenomena be reproduced late in the desquamative stage, such were, he thought, rather to be regarded as instances of reinfection, either auto-infection, as in the case related by Dr James Ritchie, or communicated by another individual. Reinfection was probably more likely to occur if the dynamic effect of the scarlatinal virus had not been fully expended in the first attack. In one of Dr

Boddie's cases the eruption was patchy in the primary seizure, while in the second the intercurrent attack of varicelle, for he had no doubt that it was really chicken-pox, favoured the theory of reinfection rather than that of relapse. He could not offer any precise statements as to the frequency with which relapses or instances of reinfection happened in the City Hospital,—probably one per. cent was not far from the truth. Epidemics of scarlet fever varied in character and intensity. In some one feature or complication was more frequent than in another—as rheumatism now, nephritis again—so possibly with respect to the proneness or otherwise to relapse or recur. He thought Dr Boddie had done good service by drawing attention to a circumstance to which too little attention had been paid. In reply to a question by Dr P. A. Young, he thought cases of reinfection were more likely to occur in hospital than in private cases.

Dr Smart, recalling his experience of the Fever House when he was in charge of it, remarked that it was then quite customary to send suspected scarlet fever cases to the Hospital, the diagnosis being at the time doubtful. The result of this practice was that many of these cases, some two or three weeks after admission, were taken with and passed through a perfectly typical attack of scarlatina. Outbreaks of this kind had far too commonly been regarded as secondary occurrences of the disease, and quoted as "relapses," or cases illustrative of "auto-infection" or "reinfection," whilst, as matter of fact, they really constituted the primary disease, the fictitious attack, on account of which the patient was sent to hospital, having been really a pseudo-scarlatinal illness. It was by no means a discredit to a medical man—not even to an expert in fevers—in the early stage of many cases, in which, as regards temperature, rash, throat, and the general condition of the patient, there was the closest mimicry of scarlatina, that he should suspend his opinion as to diagnosis. And it was his duty, moreover, where precautionary steps were so necessary, to give the patients and their relatives, as well as the community, the full benefit of any doubt which he might have. Now, with so large a number of such cases distinctly in recollection, and while fully admitting the feasibility of reinfection occurring in cases in which the original susceptibility to infection had not been wholly exhausted by a previous attack, he (Dr Smart) was nevertheless disposed to regard the percentage of such cases which actually did occur as very much smaller than was generally believed, and that such instances were only of exceptional occurrence. Then, he did not consider a "relapse" occurring in the course of a scarlatinal attack to be a relapse of the fever, or at all to be connected with an accession or renewal of the specific virus, or due in any sense to auto- or reinfection. A relapse occurring in the course of scarlet fever was brought about in the same way as in any other acute illness associated with febrile disturbance and high temperature. The

cause may, for example, proceed from an error in diet, or from a chill predisposing to rheumatic or to renal complications. The effect of the intercurrency of any of these causes, or of any other illness, was to interfere with and retard the whole machinery of metabolism, and arrest the natural operation of those processes of assimilation, secretion, and excretion, upon whose activity the normal course of the fever depended. The result was accordingly to prolong the febrile attack and delay the crisis. The Society was really much indebted to Dr Boddie for his admirable communication, and for the very practical and useful discussion to which it had given rise.

Dr Boddie thanked the members of the Society for the kindness with which they had received his paper. He was delighted to have called forth so interesting a discussion. He thought it was impossible dogmatically to fix the date at which relapse ends and a second attack takes its place: Thomas suggests four or five weeks. It was more difficult, however, to accept the statement that an attack of scarlet fever at say six weeks from the first is to be called a fresh attack, in place of a relapse. The theory of "auto-infection" which had been suggested, if it mean anything different from a relapse, seemed to him difficult of explanation. He confessed the suggestion of Henoch was more intelligible, viz., that the scarlatinal virus had not been completely eliminated, and that it may be the process of elimination had in some way been checked by some disturbance in skin, lungs, or kidneys. The suggestion that a relapse takes place where the poison has not sufficiently made its impress on the system did not appear applicable at least to his second case, especially if that impress was held to be indicated by a slight or patchy eruption.

2. A CASE OF SPONTANEOUS PNEUMO-THORAX AND PNEUMO-PERICARDIUM.

By R. A. Lundie, M.A., M.B., F.R.C.S. Ed., Edinburgh.

Pneumo-pericardium is such a rare condition, especially when it terminates in recovery, that no excuse is needed for bringing a case of it under the notice of the profession. That which I have to record presents, moreover, some features which, so far as I can discover in the literature of the subject, are absolutely unique.

The patient is a young man, clerk in a lawyer's office, aged 20 at the time of his illness. He is tall and well-made, but rather slim, and has a delicate skin and a pink and white complexion.

Family History.—The patient's parents, brothers, and sisters are alive and well. A few cases of phthisis, however, have occurred both in his father's and his maternal grandfather's family.

Personal History.—When an infant he was rather delicate, being especially liable to eczema behind the ears. When about a year old he had an acute abscess in the front of his neck, and some years later had his left shoulder dislocated. He had measles mildly; possibly scarlet fever; never whooping-cough. When about seven or eight he had symptoms, of which I can get no clear account, that the doctor who was consulted referred to hip-joint disease. The parents, unwilling to have him kept in bed, consulted a bone-setter, who ordered a blister to be applied to the hip. This was done, but in the first instance to the unaffected side; then, when the mistake was discovered, to the other; and he soon recovered. Three or four years before the illness I have to record he had on two occasions severe attacks of pain in the chest—he thinks on the left side—which he describes as quite similar to the pain which accompanied the onset of his pneumo-thorax, except that they only lasted an hour or two at most, and were not attended by the same oppression of breathing. On the day following the second of these attacks he was examined by a medical man, and understood that nothing could be discovered except palpitation. On close questioning, he stated that for two or three years he had had at times a slight cough in the morning, which brought up a spit or two; but he had been free from it for some months before his illness.

Present Attack.—On 28th April 1890, while sitting quietly at home after dinner, he felt a sudden severe pain in the region of the left shoulder-blade, which rapidly spread downwards to the axillary region, and was never again troublesome behind where it began. He had so much breathlessness and distress on the slightest exertion after this that he was forced to lie in bed, but while there felt quite comfortable. That day he had had to carry a number of heavy books at the office, and had also been swinging by his arms from a door, but felt no discomfort at the time.

On 2nd May, four days after the commencement of his illness, I was asked to see him. I found him looking perfectly well, but complaining that he had considerable pain in the left side of the chest, with breathlessness when he moved and when he lay on the left side; also that there was occasionally what he described as a "feeling of trickling" in the left side. Temperature normal; pulse 90, rather weak and compressible.

On examination, there was deficient expansion of the left chest, and the heart impulse could not be felt. On percussion, the whole of the left side was hyperresonant. The border of the hyperresonant area anteriorly passed obliquely downwards and to the right, crossing the middle line about mid-sternum, and lying considerably to the right of it opposite the lower end of the sternum. No heart-dulness could be made out anywhere. There was no change of the quality of the percussion-note in passing across the cardiac area. It was most hyperresonant from the line above de-

scribed to the mammary and upper part of the axillary regions: at the apex, in the lower part of the axillary region, and posteriorly it was less so, but everywhere much more resonant than on the right side. The breath-sounds were faintly heard in the less resonant areas, but entirely absent in the more resonant. The heart-sounds were faint, but otherwise normal, except when he had the "feeling of trickling" already referred to. During that time only there was a semi-musical accompaniment to the first sound, somewhat suggestive of the sound emitted by an elastic band suddenly stretched. No metallic tinkling was to be heard. There could be no doubt that the case was one of left pneumo-thorax. The variations in the percussion-note and breath-sounds seemed to indicate that the lung was more or less adherent to the chest wall over a rather extensive area; but the chief peculiarity was the displacement of the heart backwards instead of to the right, of which more in the sequel.

I need not enlarge upon the progress of the case at this stage. Dr Brakenridge kindly examined the patient with me on 5th May, when the condition was much the same as I have described. He remained in bed for a fortnight, his discomfort gradually diminishing *pari passu* with the physical signs, and after ten days more returned to work, apparently quite well. There was never any rise of temperature, nor interference with the appetite or digestion; and never any sign of fluid effusion.

On examining the chest after the symptoms had completely disappeared, I found that the percussion-note in the mammary and axillary regions was still slightly more resonant on the left side. Otherwise the left lung was normal. In the right there was a slightly higher-pitched note on percussion at the apex, both anteriorly and posteriorly; and in the same area there was harsh breathing, with increased vocal fremitus and resonance, but no accompaniments. The heart was normal in position and action, but was not examined with such minuteness as if I had known what was to happen later.

He continued at work till 14th June, when, while walking quietly home from his office, he felt a pain coming on in his left side, rather more gradually than on the last occasion. This time he was not aware of any unusual exertion preceding the attack. Next day, 15th June, he came to see me, and told me that he had a return of his old complaint. He stated that he had pain and discomfort in the left side of the chest, with a sensation of "splashing" there. On examining him hurriedly in a sitting posture, I found that there was not the hyperresonance on the left side which characterized his previous attack, but that the heart impulse in the fourth and fifth interspaces had a peculiar thrilling feeling, and that the heart-sounds were masked by singular irregular semi-musical accompaniments. Temperature normal; pulse 96, regular, weak. I ordered him to go home and stay in bed.

June 16.—Patient still complained of pain in the left side of the chest, aggravated by lying on the left side, and of "splashing" in the left side.

Physical examination revealed the following condition:—

Patient in Dorsal Decubitus.—No heart impulse to be seen or felt. A tympanitic area, very sharply defined, extended vertically from the second left interspace in the parasternal line to meet the stomach note, transversely from one inch to the right of the sternum to the left nipple.

Patient Sitting Up.—Over the lower part of the sternum the tympanitic note was replaced by dulness, and the heart impulse was felt in the dull area.

Patient on Right Side.—From mid-sternum to one inch to the right of the sternum dulness, and the heart impulse was felt in the dull area.

Patient on Left Side.—Dulness and heart impulse appeared within the left mamma.

Or, to sum up, there was a tympanitic area which occupied the position of a slightly enlarged pericardium, and the heart fell to the most dependent portion of it in each position of the patient. The impulse was still thrilling, as on the previous day; most markedly so when the patient was on the left side.

The sounds heard on auscultation were still more remarkable. The heart-sounds were obscured by irregular, tumultuous, tinkling accompaniments, becoming more intense with each systole. These sounds strongly suggested the splashing of a little fluid in a cavity; but their most remarkable character was their intense metallic resonance, which made the "peal of bells" almost a more apt description of them than the "water-wheel." They were most constant and intense when the patient lay on his left side, least so when he lay on his back. In the latter position they were sometimes absent, but when that was the case the heart-sounds had still a ringing metallic timbre. The patient himself was evidently conscious of the movements which produced these sounds—but by feeling, not by hearing. They were loud on auscultation, but could not be heard at a distance from the patient. Slight grating accompaniments were sometimes heard in the dorsal decubitus over the sternum about the level of the third costal cartilage. The left mammary and axillary regions were still a little more resonant on percussion than the right, but sharply contrasted with the tympanitic area over the region of the heart, and there was no evidence to show that there was air in the pleural cavity.

June 17.—Dr Brakenridge kindly saw the patient with me, and confirmed my observations and my diagnosis of pneumo-pericardium. He also pointed out that when the tympanitic area was rapidly percussed the pitch of the note changed slightly with each cardiac cycle. Unfortunately, it was not noticed at which phase it was highest. The patient expressed himself as feeling much better;

he had less pain when lying on the left side, none when lying on the back or right side.

June 18.—Tympanitic area much the same, but abnormal sounds much less in volume, and heard only when the patient lay on his left side.

June 19.—Dr G. A. Gibson kindly came to see the patient, but by this time the "splashing" felt by the patient till the previous night had disappeared, and with it the abnormal sounds. There was still more resonance than normal over the cardiac area; but in the dorsal decubitus there was relative dulness at and outside the left edge of the sternum, and the heart impulse could be seen and felt in the fourth left interspace. Dr Gibson pointed out that the stomach was distended, and that splashing could readily be produced in it.

June 24.—Præcordial region still rather more resonant than normal. Patient, feeling perfectly well, was allowed to get up for the first time. During this period, as while the pneumo-thorax was present, there was never any rise of temperature or interference with the general health. The pulse was never irregular in the least.

July 3.—Patient examined with Dr Brakenridge. There was still a little more resonance over the præcordial region than normal. The heart-sounds were normal, except that a faint murmur was heard over the sternum at the level of the third costal cartilage in the upright posture only.

May 25, 1891.—Patient was again seen. He states that he has been perfectly well, with the exception that he has occasionally what he describes as a "painful throbbing" below the left mamma, which he never had before his illness. It never lasts more than a minute, and recurs only at intervals of a week or so. He leads an active life, has been regularly doing his office work, and is now playing cricket each evening.

Physical Examination.—The right apex is in the same condition as previously noted. Resonance is now equal on the two sides at the lower part of the chest. The relative cardiac dulness begins above in the third left interspace in the parasternal line, and extends transversely from half an inch to the right of the sternum to one inch internal to the left mamma. There is no absolute dulness over the heart. The apex-beat is felt in the fifth left interspace. The heart-sounds are normal, except that in the erect posture there is a slight, short, roughish murmur heard over the sternum with the first sound at the level of the third and fourth costal cartilages, most distinct on the fourth left costal cartilage just outside the sternum; and when patient is on the right side, a longer softer murmur is sometimes, but not constantly, heard in the same position, following the first sound. Over the parts of the left lung adjacent to the heart, the respiratory murmur is interrupted. This is most distinct above the nipple and when patient is standing; it might then be described as a soft blowing murmur, synchronous with the systole, heard only during inspiration.

In connexion with such a subject as this, one is much tempted to launch into a monograph; but I must limit myself to a discussion of some of the chief points of interest raised by this particular case.

First as to the diagnosis. With regard to the patient's first seizure this presented no difficulty. The intensely tympanitic note over a great part of the left chest, with absence of breath sounds, left no room for doubt that left pneumo-thorax was present. But why was the heart displaced backwards, and not to the right?

There were none of the extraordinary sounds to be heard which characterized the second attack, and no change of the percussion-note as one passed from the normal position of the left lung across the præcordial region; so I think there can be no doubt that when I saw him there was no air in the pericardium. The pericardium must, therefore, have been displaced backwards with the heart. Now, I find that Fagge[1] states, on Fräntzel's authority, that "when there have been previous adhesions between the pericardium and the left lung, pleurisy on the left side may cause the heart to be carried backwards away from the chest wall, so that no impulse whatever can be felt or seen." If this can happen with fluid effusion, then it should take place still more readily when air has entered the pleural cavity, and the whole weight of the heart (in the dorsal decubitus) can aid the traction exercised by the lung. There was evidence of adhesion of the pleura in various other situations in this case, which lends plausibility to the hypothesis that there may have been adhesion also to the pericardium. It is remarkable, too, that the pain first experienced by the patient was in the region of the left shoulder-blade, where there was comparatively little physical evidence of pneumo-thorax; while it quickly passed to the axillary region, where it remained almost exclusively during the subsequent progress of the case. "Pain in and about the left shoulder" is mentioned as an occasional symptom of pericarditis;[2] and it is not difficult to believe that a sudden strain on the pericardium posteriorly might produce the same symptom, and that it might pass off when the tension was relieved as the air displaced lung and pericardium backwards together. I believe, therefore, that in my patient there was adhesion between the pericardium and the left lung, and, especially in the light of subsequent events, that this was the situation in which the rupture of lung occurred which led to the pneumo-thorax. Moreover, I think the curious accompaniment to the first sound, heard sometimes while pneumo-thorax was present, and the slight murmurs still present in certain positions of the patient, may best be explained as due to slight constraint of the heart, produced by traction on the pleuro-pericardial adhesions.

[1] *Princ. and Pract. of Med.*, second edition, vol. ii. p. 168.
[2] Sir T. Watson, *Lectures*, fifth edition, vol. ii. p. 329.

In the second attack the diagnosis was equally easy; all the typical signs described by writers on pneumo-pericardium were present with exquisite distinctness. The signs afforded by percussion and palpation were in themselves sufficient; a cavity containing air, occupying the precise situation of the pericardium, in which the heart changed its position freely from side to side, and from back to front, with alteration of the patient's posture, could be nothing but the pericardium itself. And when, in addition, there was the "water-wheel sound," which has generally been regarded as pathognomonic of hydropneumo-pericardium, the evidence was overwhelming.[1] The general impression conveyed by the sounds heard was that there was a splashing of fluid, but of a very small quantity: probably a teaspoonful, much less than the average amount found after death, would be sufficient to produce the effects observed. There was not the extreme loudness of the sounds described by some writers in connexion with the condition; but this, though a most striking feature when it does occur, is only occasionally present.

When we enter upon a consideration of the pathology of the case, however, things are much less clear. I think there can be no doubt that the two attacks must be closely associated with each other. I believe that in all probability a rupture of the lung into the pleural cavity occurred close to adhesions to the pericardium; and that close to the first rupture, possibly in consequence of the contraction of its cicatrix, a second rupture occurred into the pericardium itself. What the lesion which caused the first rupture may have been must be purely a matter of speculation. The explanation which most naturally suggests itself is the presence of an ulcerating phthisical cavity close to the pericardium, which ruptured first into the pleural, then into the pericardial cavity; but this does not seem to me to tally with the facts of the case. Of course, many cases of pneumo-thorax occurring in the early stage of phthisis do extremely well; but this is usually attributed to the physiological rest given to the affected lung-tissue, and to the pleura in its neighbourhood, into which infective elements are likely to escape. In pneumo-pericardium the conditions are almost the contrary; any elements which escaped into the pericardial cavity would be churned up in the fluid present by the movements of the heart, and the phthisical lung-tissue, supposing such to be present in contact with the pericardium, even if not subjected to unusual disturbance, as seems likely, would certainly not be quiescent, as in the

[1] It has been shown, as I think satisfactorily, by Reynier* that the "water-wheel sound" may occur when the air and fluid occupy, not the pericardium, but the loose areolar tissue between it and the sternum; but this could hardly occur without subcutaneous emphysema as well, and in any case would not give the beautifully definite percussion signs presented by my case. This is the only condition besides hydropneumo-pericardium, so far as I can discover, which has been *actually observed* to produce the sound.

* *Arch. Gen. de Med.*, 1880, vol. i. pp. 441, 582, *sq.*

case of pneumo-thorax. On this supposition, therefore, the absolute freedom from any symptoms of inflammation of the serous membranes, not only for some months before, but during and ever since the illness, seems to me hardly conceivable.

It seems probable, however, that the adhesions I believe to be present are the remains of an old insidious pleurisy, and this receives confirmation from the very remarkable description given by the patient of severe, though transient, pain in the chest some years ago. Possibly an unusual effort may have strained old adhesions at one place to the point of rupture. Whatever the lesion may have been, I think we are fully warranted in concluding that the normal structure of the serous membranes in this patient must be extremely delicate. The pericardium is generally, as we know, although not a thick, yet an extremely tough and resistent fibrous membrane. There are, however, great variations in this respect. Dr Bruce tells me he has met with a pericardium which appeared to consist of hardly more than endothelium; and if we suppose such a condition present in my patient, the difficulty of imagining the mechanism of a perforation of the membrane is greatly diminished.

In comparing my case with others on record, the most remarkable feature is the entire absence of any signs of other serious disease, or of any symptoms, except the comparatively slight ones which could be accounted for by the mechanical presence of air. The two cases which come nearest to mine in this respect are that reported by Soraner[1] from Traube's clinic, in which there was only slight fever and slight rheumatic pericarditis; and Love's[2] case, in which, however, the diagnosis seems to me by no means convincingly established, where there was also only slight fever, with slight friction in the region of the heart, possibly both pleural and pericarditic.

It is, of course, possible that there may have been pleurisy or pericarditis in the region where I believe the ruptures to have occurred, quite undiscoverable by physical examination. But if so, it must have remained singularly slight, and extremely localized.

A word as to prognosis. This case goes to confirm the less sombre view that has of late years been taken of pneumo-pericardium. There seems now to be no reason to regard the presence of air in the pericardium *per se* as a very alarming condition. All depends upon the route by which it has found entrance, and the other lesions with which it is associated. In traumatic cases, where there are no other injuries, a large proportion recover.

[1] "Diss. Inaug. de Hydropneumopericardio." Berlin, 1858. See Canstatt's *Jahresbericht*, 1858, 3, p. 215.

[2] *Lancet*, 1888, vol. i. p. 319. It is remarkable that this is the only case of pneumo-pericardium I can discover recorded from the United Kingdom since Warburton Begbie's, in the *Edinburgh Medical Journal* for 1862.

When the primary disease is pericarditis, as in many of the non-traumatic cases, a certain number do well. But I have failed to find an instance of recovery when the pericardial cavity has communicated, either from injury or disease, with the œsophagus or stomach.

No *treatment* was adopted in this case except rest in bed and tonics; and there was no indication whatever for anything more.

In conclusion, I shall enumerate the most remarkable points in the history of the case, by way of summary.

1. The presence of a chronic morbid process in the chest, sufficient to lead ultimately to rupture of the pleura and pericardium, with an almost entire absence of symptoms.
2. The occurrence of pneumo-thorax in conditions of apparent health.
3. Backward displacement of the heart while pneumo-thorax was present.
4. Recovery from pneumo-thorax without any signs of inflammation or effusion.
5. The occurrence of pneumo-pericardium after an interval, also in conditions of apparent health.
6. The entire absence of irregularity of the pulse, disturbance of the circulation, or interference with the general health while air was present in the pericardium.
7. Recovery from pneumo-pericardium without any signs of inflammation or effusion, except the small, perhaps normal, amount of fluid in the pericardium indicated by the auscultatory signs.

Dr James expressed the very great interest with which he had listened to this most instructive and interesting paper, and stated that he thoroughly agreed with Dr Lundie in his explanation of the sequence of events. Pneumo-thorax had occurred, the usual displacement of the heart to the right had been prevented by adhesions between the pericardium and lung, and through the pericardium rupture had occurred, so causing pneumo-pericardium. The only case in any way resembling it which he could recollect of was one quoted by the late Warburton Begbie in his article in Reynold's *System of Medicine*. In this case the mischief was tubercular, and in Dr Lundie's case it seemed to him that the process was also tubercular. At the upper part of the left lung tubercular deposit had been existent, causing adhesions between it and the pericardium, and also at parts between it and the chest wall. Pneumo-thorax had occurred in the ordinary way, and the strain on the adhesions between the lung and pericardium had caused a rupture through them into the pericardium, and escape of air into it from the pneumo-thorax. The fact that both the pneumo-thorax and pneumo-pericardium was followed by little or no pleurisy or pericarditis and rapidly healed, was in favour, he thought, of their tubercular origin, at any rate he had seen and heard of cases of early

phthisis in which the occurrence of pneumo-thorax not only produced no pleuritic effusion, but seemed, by giving rest to the lung and allowing cicatrization, to heal the cavity which had caused it.

Dr Smart, alluding to the great clinical ability displayed by Dr Lundie in the diagnosis and management of his most interesting case, and to the masterly way he had brought the case before the Society, went on to cite the case of a young invalided soldier who died in Ward VI., and whose case bore some resemblances to Dr Lundie's patient. The autopsy revealed the existence of adhesions between the pericardium and pulmonary pleura, which had ended in perforation leading to pneumo-thorax, and which would have also led to pneumo-pericardium had not the pericardium become adherent to the heart. By means of these adhesions the collapsing lung had pulled the heart upwards and backwards under the left clavicle, so that its apex beat was in that quarter. The collapsed lung, itself honeycombed with tuberculous ulcers, was lying against the back of the left thorax. There was no effusion. This, and similar cases which he had seen, led him to think that Dr Lundie's case was of tubercular origin, and would probably run a similar course. He (Dr Smart) incidentally mentioned, in connexion with the case, that the title of one of his papers on undescribed neuroses, and which he trusted to have the privilege of bringing before the Society, was Pseudo-Pneumothorax, the object of which was to show that many published cases of so-called pneumothorax were fictitious and misnamed, having their origin in an entirely different condition. Dr Smart referred to diagrams of respiratory tracings in explanation of his views.

Dr Lundie, in reply, said that he had not stated that there was no tubercular (or, as he would prefer to call it, phthisical) process *at the bottom* of the mischief in his case, but that he thought an *ulcerating phthisical cavity* was excluded by the circumstances. He was well aware of the fact insisted upon by Dr James and Dr Smart —that early cases of phthisis in which pneumo-thorax occurs from rupture of a small cavity often do well; but that this should occur first in a pneumo-thorax and then in a pneumo-pericardium, where the conditions must be in some respects precisely the opposite, seemed to him so very improbable that he preferred to lay stress on the probable presence of extremely delicate tissues in his patient as being the most important factor in determining the ruptures.

Meeting X.—June 17, 1891.

Professor A. R. Simpson, *President, in the Chair.*

I. Exhibition of Patients.

1. *Dr John Thomson* showed two children with chronic cerebral lesions. (1.) A little girl with a *lesion near the floor of the 4th ventricle.* The patient was 2 years old, and had been sent to the New Town Dispensary by Dr E. E. Maddox. Her symptoms were—(*a*) complete paralysis of both external recti, with very extreme double internal strabismus; (*b*), paralysis of both sides of the face, rather less marked on the left than on the right side, and on both sides showing this peculiarity—that while the muscles of the lips, cheeks, and forehead were almost absolutely immobile, the eyelids could be tolerably well closed, the orbicularis palpebrarum being comparatively little affected; (*c*), a curious inability to open her mouth, even when her pharynx was tickled she was unable to separate the front teeth more than two-thirds of an inch; (*d*), extreme irritability dating from onset of other symptoms. The child was normal in other respects, with the exception of a chronic catarrhal condition of the nasal mucous membrane and also presumably of the pharynx. The muscular power of the arms, legs, and body was good, and the eye-muscles (with the exception of the external recti) were normal. Cutaneous sensibility, sight, and hearing were unaffected. The larynx and palate were probably normal. The movements of the transverse muscular fibres of the tongue seemed impaired, but its other movements were pretty free. The intelligence was normal; there was no headache and no vomiting. The urine contained neither sugar nor albumen. As to the onset and progress of the symptoms—the right eye was noticed to turn in suddenly during the last week of last December, the child being very well at the time; the left eye became similarly affected between a week and a fortnight later, and the irritability developed soon after. The paralysis of the right side of the face was first observed in February. The inability to open the mouth appeared about six or seven weeks ago, and at that time was so extreme that the teeth could only be separated about one-fourth of an inch, and there was great difficulty in feeding the child. For the last fortnight iodide of potash had been given, and there had been slight general improvement. The nature of the lesion was very obscure; there was no history of syphilis. The seat of the lesion was evidently near the floor of the 4th ventricle, and it implicated the nuclei of both 6th nerves and also those of the 7th nerves, or else those nerves themselves in the neighbourhood of the former nuclei. The inability to open the mouth was probably to be accounted for by supposing some implication of the adjacent nuclei of the muscular portion of the 5th nerve. (2.) A child, aged 6, *recover-*

ing from a tumour in the region of the right crus cerebri. Fifteen months before, she had been shown to the Society. At that time she had had—(*a*), paresis of left side of the face and of the left arm and leg; (*b*), complete paralysis of the muscles supplied by the right 3rd nerve; (*c*), slight optic neuritis; (*d*), marked mental dulness. Now, the paresis of the left side was considerably less; slight dilatation and sluggishness of the right pupil was all that was left of the affection of the 3rd nerve on that side; the optic neuritis had long ago disappeared, and the child's mental capacity and alertness had almost entirely recovered, so that she took a good place in school and was good at going messages. The treatment had consisted in careful feeding, cod-liver oil, and iodide of iron. The child had gained very much in weight and in general strength. The lesion was regarded as probably of the nature of a caseous mass.

2. *Dr Alex. Bruce* showed two cases of FACIAL AND OTHER NERVE PARALYSIS.

3. *Mr. A. G. Miller* showed—(1.) A case of HÆMOPHILIA with perfect family chart for three generations, showing males all bleeders, and transmission through females only. Patient (boy of 13) had effusion into right knee-joint six times from slight injuries. Right leg shows an elongation of about an inch, which is due to increase in length of both femur and tibia. (2.) Two cases of EXCISION OF WRIST by Dr Heron Watson's method. One operated on a year ago, with useful hand resulting. Other operated on a month ago, with good movement of fingers. Also bones shown removed from a case operated on the day before. Essential peculiarity of Dr Heron Watson's method is a single ulnar incision, by which no tendon or vessel requiring to be ligatured is cut. (3.) Case of ABSCESS OF THE ANTRUM cured in two months by removal of anterior molar and free irrigation through enlarged socket of tooth. Recurrence of discharge from nose after six months. Frontal abscess diagnosed, and frontal sinus opened. Free irrigation into nose established a cure in two weeks. (4.) Photograph of CHILD on whom laparotomy was performed a year ago for tubercular peritonitis. Child reported perfectly well and attending school regularly.

4. *Dr Andrew Smart* showed a case of critical ALCOHOLIC SINGULTUS, continuous for weeks; recovery; treatment. The patient, a navvy, was brought by the police to Ward VI. on 20th May. His condition was that of collapse, with gravescent coma and continuous singultus; the hiccough, of a distressing and exhausting character, recurring sixteen times a minute. It appeared that, on account of this condition having continued three weeks, the patient had not been able to obtain sleep, or take nourishment, however little, by the mouth without aggravating his trouble. It was accordingly deemed necessary from the outset to suspend absolutely feeding by the stomach, having recourse to nutrient suppositories

and enemata; and, with the object of ameliorating the gastric neurosis, morphia—in this case sulphate—was administered hypodermically every three hours, one-third of a grain each time. To permit of this strength of morphia being continued sufficiently long, and to guard against the occurrence of toxic narcotism, chloroform was administered simultaneously with the morphia, either by inhalation or by ingestion—in the latter case 60 minims of the *spirit* of chloroform being given each time. When inhalation was used it was found necessary to continue it until it arrested hiccough, and this usually did not occur before chloroform narcosis was induced. We succeeded, by these methods, in arresting the continuity of the singultus, and after forty-eight hours continuance, day and night, the patient had gained seven hours' sleep. By the sixth day diaphragmatic spasm was completely arrested, and, by the ninth, feeding by the mouth was gradually resumed, the patient thereafter progressing towards recovery. For twelve days the patient was absolutely fed by rectum. This method, during the whole course of the case, was well tolerated by the bowel, and so carefully carried out by the nurses, in reference to details, as to have been rendered perfectly successful. The case is instructive as representing the severe and usually fatal type of singultus connected with chronic alcoholism.

5. *Mr Caird* showed a case in which the METACARPAL OF THE THUMB HAD BEEN REMOVED by Mr Chiene for epithelioma.

6. *Dr Alex. James* showed a patient with DISPLACEMENT OF THE HEART due to lung disease. This patient, a man aged 44, had been rowing in a boat race nine years previously, when he states he felt "something give way" in his right side, and at once became so faint and weak that he had to be carried home. Next day he coughed up a large quantity of blood. In the course of a few weeks he recovered; but he states that he has had a cough, and once a year an attack of hæmoptysis, ever since. This cough and hæmoptysis have been slight, and his general health has been perfectly good until about a year ago, when he had an attack of influenza. Since then the cough has been much worse, he has been losing flesh, and he has suffered from back pain and increased frequency of micturition. At present physical examination shows, with slight supra-clavicular hollowing, extensive fibroid phthisis of the right upper lobe, and a small quantity of pus and albumen in the urine. The diagnosis is, therefore, chronic tubercular disease of the right lung and probably slight renal pelvic or prostatic tubercle. The peculiarity is, however, the very great displacement of the heart to the right. Pulsation is visible in the 2nd, 3rd, and 4th interspaces to the right of the sternum. The locality at which the greatest displacement of the heart to the right can be recognised is the 4th interspace. Here pulsation can be felt, and percussion dulness of the heart can be elicited at the right nipple line.

The left lung is in a condition of hypertrophous emphysema, and a resonant note is elicited by percussion over the normal cardiac area to the left of the sternum. This patient was sent to me at the Infirmary waiting-room as an example of "dextracardia" in which none of the other viscera were transposed. The condition is of course not congenital, but due to the lung disease. Although of course some cardiac displacement is a usual associate of all chronic phthisical lung disease, this is the third example of excessive displacement which I have met with. In all of these there was a history of rack or strain.

7. *Mr Caird* showed—(1.) A case of EXCISION OF EXOPHTHALMIC GOITRE. The patient, W. N., æt. 25, came to the Edinburgh Royal Infirmary from a limestone district in the North of England, where goitre is prevalent. He had suffered from a swelling in the neck for four years. Of late it had grown with rapidity. He suffered from palpitation, dyspnœa, and dysphagia. There was a large cystic goitre which extended from the hyoid to beneath the manubrium sterni, and which had forced the trachea and œsophagus to the left of the middle line. The eyes were markedly protruding, the pulse-rate very frequent (116 to 124). Excision of the large cyst and right thyroid lobe was performed after Kocher's method. The stitches were removed on the 7th day, and the patient got out of bed on the 10th day. A year has passed since the operation, and the patient has since enjoyed perfect health. The eyes no longer protrude, the pulse is less frequent, there is no longer any discomfort or dyspnœa. (2.) A case of EPITHELIAL GRAFTING after Thiersch's method. The patient had an extensive granulating area from above the external malleolus over the dorsum of the foot and under the sole. It had been caused by molten iron. Grafting in two sittings had healed the whole in 14 days.

II. Exhibition of Specimens.

1. *Dr William Russell* showed casts illustrating diseases of the LUNGS AND LIVER.

2. *Mr Caird* showed two ABDOMINAL ANEURISMS.

Meeting XI.—July 8, 1891.

Professor A. R. Simpson, *President, in the Chair.*

I. Exhibition of Patient.

Dr Smart showed a patient, a woman 50 years of age, exemplifying complete recovery from SEVERE DIABETES MELLITUS after eight years' continuance. When first seen by Dr Smart she was

eliminating nearly 12,000 grains of sugar daily, the urine being over 200 ounces daily. The cure of her disease was effected within a year from the commencement of treatment, and has not, after seven years, returned, although during that period she has been taking absolutely unrestricted diet.

II. Exhibition of Specimens.

1. *Dr W. Russell* showed a KIDNEY with a large sarcomatous growth in it, and with rounded projecting growths on the pelvis and calyces, which were covered by urinary salts, giving them a mulberry appearance, and closely resembling calculi.

2. *Dr Alexis Thomson* showed for Dr Harvey Littlejohn two cases of RUPTURE OF THE GUT FROM VIOLENCE, and a case of INTUSSUSCEPTION OF THE BOWEL:—

(1.) *Ruptured Intestine due to Violence.*—Woman, aged 40, quarrelled with her husband on Saturday night, and is said to have received a kick on abdomen. Complained of pain in abdomen, and illness all Sunday, and died on Sunday night, was able to go about on Sunday, and never accused her husband of kicking her.

P. M.—No external marks of violence, but on opening abdomen an extravasation of blood about size of an orange was found in and under the peritoneum to the left, and below the umbilicus. Intense peritonitis, recent yellow lymph matting the intestines together. The omentum was fixed by a small adhesion near inguinal ring. There was a tear in the omentum with mass of clotted blood; below this (after reflecting omentum) was found a rent in the mesentery with ragged edges, and of uneven shape, coated with lymph. On removing this a coil of intestine lying against the vertebral column was found covered with lymph, under which the bowel was ruptured in *two places*. The injuries in the omentum, mesentery, and gut were all in a direct line from bruise on peritoneum.

Points to be noted.—1. No external injury. 2. Power to move about house, and take food after the injury on Saturday night. 3. Triple injury to:—(1) omentum, (2) mesentery, (3) gut. 4. Character of injury to gut, viz., double rupture. 5. Did a kick, or could a fall have done it?

(2.) *Ruptured Intestine due to Violence.*—Man while at work with a circular wood saw received a severe blow in right iliac fossa from plank of wood. Walked home; went to bed ill and vomiting. No fever, great bruising of right iliac fossa, hip and thigh. Died in forty-eight hours.

P. M.—Peritonitis more or less general, but no lymph effused as in former case. Great number of small hæmorrhages on surface of the intestines. A rupture was found in the ileum, which was peculiar from the fact that it resembled very closely an ulcer,—this

appearance, however, being due to the edges being folded back, and held down by adhesions, and to the circular shape of the rupture.

(3.) Shows very well the appearance of the bowel in *Intussusception.*—The history obtained is the following:—A girl aged eight years returned home from school complaining of a sore belly. Nothing was done at first. Later she was taken to a chemist where she got a dose of castor oil. This apparently doing no good she was taken to another chemist, who administered a dose of jalap. Severe vomiting and diarrhœa set in, and she died in three days.

P. M.—Abdomen distended. On opening abdomen all intestines were seen to be of deep plum colour. No peritonitis. *Intussusception* found high up in jejunum. It felt like a bag of porridge, and presented a somewhat circular appearance, the two extremities being approximated.

Points to be noted.—1. No peritonitis; 2. All intestines full of plum-coloured mucus; 3. Intussusception high up in jejunum; 4. Medico-legal relations *re* responsibility of parents and chemist.

3. *Dr John Thomson* showed a portion of SMALL INTESTINE showing a *Meckel's Diverticulum,* from a child aged ten months. The diverticulum was 1¼ inch in length, and was quite free at its extremity, which was slightly bifurcated. It was situated nearly 30 inches above the ileo-cæcal valve.

III. Original Communications.

1. ON THE FUNCTION OF THE TONSILS.

By G. Lovell Gulland, M.D., F.R.C.P. Ed.

Before considering the function of the tonsils, it may be well briefly to recapitulate their structure, as in some of the more recent papers on this subject erroneous statements have been made.

The essentials to a tonsil are—a fold, ingrowth, or invagination of epithelium whose lumen is still continuous with that of the alimentary canal, and, surrounding this epithelial pit, a mass of adenoid tissue, whose presence may or may not cause a projection on the free surface. This tissue consists of a supporting framework of connective tissue, carrying numerous bloodvessels and lymphatics; in the meshes of this framework lie leucocytes in various stages of development. At some points the arrangement of connective tissue and bloodvessels may be so altered as to give rise to those structures which in this country are generally known as "follicles;" but which, as they have no relation to glandular follicles, it would be better to call "germ-centres," as Flemming (8)[1] has proposed to do.

[1] The numbers refer to the bibliography at the end of the article.

In some cases the tonsil consists of a single pit or invagination of this sort,—as, for instance, in the faucial tonsils of the rabbit and cat, and in the individual parts of the lingual tonsil in man; but more usually a number of these simple tonsils lie so close together that their adenoid tissue becomes continuous, or secondary epithelial crypts surrounded by adenoid tissue spring from the primary crypt, and a compound organ is thus formed. These processes often occur simultaneously,—as for instance, in the faucial tonsil of man,—and the appearance of such a compound tonsil on section is that of a number of irregular crypts bordered by epithelium and imbedded in a diffuse adenoid infiltration, in which are germ-centres in varying number.

The epithelium of the tonsils is, of course, continuous with that of the mucous membrane around, and is therefore a stratified squamous epithelium in the palatal, lingual, and part of the pharyngeal tonsils, while over the upper and anterior part of the pharyngeal tonsil it is generally ciliated (*cf.* Suchannek, 22, Schwabach, 21, Killian, 18). The tonsillar epithelium in all these situations differs, however, very markedly from that of the rest of the mouth and pharynx, inasmuch as its continuity is broken by the emigration through it of enormous numbers of leucocytes. These are produced in the adenoid tissue beneath, wander between the individual epithelial cells, or through wide channels formed by the destruction of the epithelium by the previous passage of leucocytes, enter the lumen of the crypt, and so pass on into the cavity of the pharynx and mouth. In the latter situation the emigrated leucocytes are known as "salivary corpuscles." Stöhr (3) was the first to show that this emigration is a normal occurrence, and in a series of papers (6, 7, 11, 13, 14, 25) he has demonstrated that in the conjunctiva, nasal mucous membrane, urinary bladder, small intestine, and, in short, wherever an accumulation of leucocytes underlies epithelium, the same emigration takes place. I think it the more necessary to lay stress upon this fact in connexion with the tonsils, because the most recent writer on their structure, Hodenpyl (29), has misunderstood the process, and considers that this "rarefaction of the epithelium," as he calls it, is the result of pressure from beneath, and that it "facilitates the passage of lymphoid cells from the tonsil to the cavity of the mouth." As a matter of fact, the leucocytes actually destroy the epithelial cells, and it will be found that the destruction is most nearly complete, and the passages through the epithelium are widest just above the germ centres, where the constant attack of fresh leucocytes prevents repair of the epithelial covering.

The connective tissue of the tonsils does not require special description. It consists of a network of fine fibres, interlacing in every direction, so as to produce the very fine meshes in which the leucocytes are detained.

The ultimate distribution of the bloodvessels was first worked

out by Schmidt (1), whose Taf. XVI. shows their arrangement excellently. This is in so far unusual, that the arteries do not penetrate far into the tonsil, but soon break up into capillary vessels, often of quite unusual length. The veins are very numerous, of very large calibre, and have very thin walls,—altogether the conditions, as will at once be perceived, are specially favourable for the emigration of leucocytes. The lymphatic vessels commence in the tonsils by open radicles in the connective tissue spaces. Retterer (19) doubts this, but Schmidt (1) had previously quite definitely proved it, and had given an excellent schematic figure of the arrangement (Taf. XVI. 6), and with complete series of sections, it is quite easy to trace the connexion between connective tissue spaces and lymph vessel. The lymphatics unite into several large trunks just as they leave the tonsil; and these trunks, crammed with leucocytes, can easily be distinguished in the peritonsillar connective tissue. There are no lymphatics passing to the tonsil.

The "germ-centres" require a somewhat more detailed description, as their structure and function are not understood by most of the writers on the subject; and Hodenpyl in particular is in error in considering that no difference can be detected between the cells in the "nodules," as he calls them, and the adenoid tissue around. These structures are parts of the tonsil (and of adenoid tissue elsewhere), specially differentiated in order to further the mitotic division of leucocytes; and it was by the use of a special fixing solution and of pure nuclear stains that Flemming (8) first showed their true character. They have the appearance on section of round clear spots surrounded by a dark ring, and in the tonsil are arranged radially round the crypts. The prime cause of their formation is, I think, to be sought in the bloodvessels. Swain (12), though he has erroneous ideas about the structure of the germ-centres, points out that the capsule of the tonsil is produced by the fact that the leucocytes emigrating from the vessels cause an increase of bulk in the connective tissue at that point, and that as a consequence the connective tissue surrounding the infiltration is compressed, and thus apparently forms a capsule. The same thing occurs in the germ-centres; they have a peculiar arrangement of bloodvessels, from which their other peculiarities of structure result. Only capillaries enter the germ-centre, and these pass in, in a radially centripetal manner, curve on themselves in the centre, and pass out again to enter the venule. This disposition, of course, results in the massing of terminal capillary loops at the centre; and, therefore, while the current of blood in the afferent capillaries is radially centripetal, the pressure outside the capillaries, produced by the *vis a tergo* of exudation, will be radially centrifugal. Further, the blood-supply to any cells lying in the germ-centre will, evidently, be very abundant. The meshes of the connective tissue supporting these capillaries are very fine, and the fibres are

very delicate. In the germ-centre the reticulum is disposed irregularly, but at its margin is condensed, and the fibres are so drawn out that the meshes run at right angles to the radius of the sphere of the germ-centre, and the fibres form a sort of capsule to the germ-centre, produced by centrifugal pressure, just as the capsule of peritonsillar connective tissue is formed (see Hodenpyl's fig. 2). Outside this capsule the fibres become continuous with those of the ordinary adenoid tissue; but it is to be noted that there are always many lymphatic vessels just outside the capsule ready to receive any cells which pass through it (*cf.* Schmidt, Taf. XVI. fig. 3). There are no lymphatic vessels inside the germ-centres, nor where germ-centres underlie epithelium are there any on the epithelial side of the germ-centre.

From this description it will be understood that any leucocytes which pass out of the capillaries in the germ-centre will be held for some time in the fine meshes of the connective tissue in the interior, but will always be pushed on slowly towards the periphery; when they reach the close meshes at the margin they will be detained still further, but ultimately the pressure from within will drive them through the meshes and force them either into the lymph vessels, into the diffuse adenoid infiltration surrounding the germ-centre, or through the epithelium. The pale appearance of the germ-centre on section when a nuclear stain is used is due to the fact that the leucocytes contained in it are principally large resting cells about to divide, as I have elsewhere described (27), which have a "vesicular" nucleus, to use an obsolete but expressive term. These divide rapidly, by mitosis, and the daughter-cells, which are small cells whose nucleus has a dense chromatin network, are carried much more quickly through the connective tissue meshes of the germ-centre than the large leucocytes of whose division they are the product, and when they are detained for a time by the closer meshes at the periphery, give rise to the deeply-stained ring of closely packed small cells which is seen there in sections. When these young leucocytes finally pass out of the germ-centre they develop according to the circumstances in which they are placed, either into the wandering leucocytes with lobate or polymorphous nuclei, or into the stationary leucocytes with "vesicular" nuclei and large cell-body, the largest of which are the well-known "macrophages." Leucocytes seem to develop into the former stage when food is scanty or has to be sought for, into the latter form where it is abundant and to be had for the taking. Most of the leucocytes in the tonsils outside the germ-centres are of the stationary stage in different stages of development, the largest macrophages lie generally just under the epithelium. Wandering leucocytes are rarely found in these organs except in the bloodvessels or lymphatics, or making their way through the epithelium. The reason for this superabundance of the stationary stage is probably

to be found in the very free blood-supply of the tonsil. The leucocytes in the mucus covering the surface of the tonsil are almost invariably in the wandering stage, as one would expect.

The development of the tonsils I have described elsewhere (28).

These are the main facts in regard to the structure of the tonsils, and we may now pass to consider their function. We should have been spared much vain theorizing on this subject if writers on the human tonsil had examined the same organ in other animals, and I may note in this connexion that either faucial, lingual, or pharyngeal tonsils are found very widely in mammals (*cf.* Schmidt, 1, Killian, 18, Retterer, 19), that they are found also in birds (Killian), while Holl has described tonsils in the buccal cavities of the salamander (10) and frog (16), and Oppel has found them in the pharynx of Proteus anguinus (24). A study of the characteristics of the tonsil through the animal series completely negatives the idea that this structure is a functionless survival of some pre-existing organ. The tonsils of the frog, salamander, etc., are single shallow epithelial pits with a slight leucocyte infiltration round them, very like the first stages in the development of the tonsil in mammals, while as we advance through the scale to the Primates, the increase of the tonsils in number and in size, both relative and absolute, the increasing number and complexity of the crypts, the appearance of germ-centres, and the increased activity of leucocyte proliferation, all show that the tonsils have some very real and active function to perform.

Hodenpyl (*l. c.*) has discussed and disposed of the older theories of the functions of the tonsils, and I need not here examine them. The more recent theories, however, are those of Hingston Fox (15), Spicer (20), Hill (17), and of Hodenpyl himself.

Hingston Fox imagines that the buccal fluids accumulate in the crypts, pass through the mucous membrane, and are thus absorbed, while Scanes Spicer makes the extraordinary suggestion that the pharyngeal tonsil is meant to absorb the nasal and lachrymal secretions when one is lying down, while the lingual tonsil does the same when one is erect, as the uvula drips on to it in that position! The faucial tonsils are supposed to do something of the same sort, and the whole of them are expected to act as "sewage farms" for the contaminated nasal and buccal secretions. Hill also believes the tonsils to be absorbent organs, but thinks that they have to deal with the products of salivary digestion and the buccal secretions, both of which are supposed to be absorbed by the buccal and faucial mucous membrane, and to pass thence into small lymphatics which go to the tonsils. This is anatomically impossible, as there are no lymphatic vessels passing to the tonsil in this manner; these organs are peripheral lymph-glands, not intermediate ones. Baumgarten (4) also regards them as absorbents, because he found tubercular ulcers on them in tuberculosis produced by feeding. The difference between the absorption of

food and that of microbes is considerable, however, and Hodenpyl, who examined the tonsils of 200 persons for tubercles, found them only in one case, where the individual had "died of very general tuberculosis." This seems to indicate that Baumgarten's results are capable of another explanation. Hodenpyl has, moreover, made a series of very careful experiments, in the course of which he applied olive oil, melted lard, lanolin, carmine, Berlin blue, emery powder, aniline colours, salts of iron, and solution of atropine to the surface of the tonsils, and in no case did absorption of any one of these take place to any appreciable extent.

There is no reason to regard the tonsils as concerned in the absorption of nutriment. The foods we use can only (for the most part) be absorbed after digestion, and the bolus of food before it passes the tonsils has been subjected to no digestion whatever. There is, it is true, an amylolytic ferment in the buccal fluid, but the food does not stay long enough in the mouth to have its starch converted into sugar, and, moreover, what is to be made of the case of carnivorous animals, whose normal food contains no starch, and which yet have large tonsils? No constituent of their food is digested in the mouth, and yet their tonsils are evidently of use to them. The contact of the food with the tonsils, moreover, is only a momentary one, as the bolus is hurried through the fauces. Fox (*l. c.*) makes the suggestion that some part of the food is pressed into the tonsillar crypts during the act of swallowing, but while in the human tonsil some of the crypts open towards the front of the mouth, and this might conceivably happen to a very limited extent, in most of the animals with simple tonsils, as in the rabbit, cat, and guinea-pig, the single crypt opens towards the posterior aspect and is protected from the entrance of food by an overhanging anterior lip. Moreover, the stream from the tonsils is in an outward direction, and the crypts are normally full of leucocytes and mucus, which are passing out into the cavity of the mouth. None of the observers I have mentioned were acquainted, apparently, with Flemming's or Stöhr's work, so that their theories could be of little value.

Still further objection to any theory which assumes that the tonsils have to do with absorption of food is to be found in the consideration, that even if the faucial tonsils were occupied in this way, we should still be unable on that hypothesis to explain the function of the pharyngeal tonsil, with which food never normally comes in contact. Now, the structure of all the tonsils is practically identical as far as essentials go, and we are therefore entitled to assume that their functions must also be the same. The outstanding fact in regard to them all is, then, that the tonsils are arrangements of bloodvessels and connective tissue for the purpose of furthering the division of leucocytes. The germ-centres where this production of new leucocytes mainly takes place are so situated that the young cells pass from them on one side into the

lymphatic system, and so ultimately into the blood, by which they are distributed all over the body, and on the other side through the epithelium into the tonsillar crypts, and so into the alimentary and respiratory passages. The function of the tonsils is then to produce leucocytes. We must now inquire in what way the leucocytes can be utilized in this particular situation. Leucocytes, as I have elsewhere maintained (27), are to be regarded as unicellular organisms which have the characteristics of Protozoa,—the power of amœboid movement, the power of ingesting foreign matters by that movement, and of utilizing these particles for nutrition when they are capable of being so utilized. They have some other peculiarities which in this connexion are not of special importance. Possibly, for instance, they are concerned in the transport of nutriment from the absorbing surfaces to the tissues, as Hofmeister maintains (9); but as I have shown that the tonsils cannot be concerned in absorption, we may leave that point out of consideration here, though of course the leucocytes which pass into the lymphatics may ultimately come to be employed in that way, and the tonsils may thus indirectly be implicated in the function of nutrition.

The characteristics of leucocytes which I have enumerated above are those which specially fit them to "protect" the other cells of the organism, to act as "phagocytes," if I may be allowed to use that word without committing myself altogether to the views of Metschnikoff and his school as to the relations between leucocytes and microbes. It will be seen immediately, that if we consider the position of the various tonsils and various other facts which I shall adduce, we can hardly avoid the conclusion that the tonsils are "protective" organs. At the back of the human mouth and upper part of the pharynx there are two rings of adenoid tissue (*cf.* Bickel, 5). The lower of these is the one surrounding the fauces, and is formed of the lingual tonsil below, the palatal tonsils on either side, and above of the slight adenoid infiltration which Bickel describes as running along the lower surface of the velum palati. If the pharynx is opened from behind, and the parts adjusted in the position which they occupy in life when the mouth is shut, it will be seen that the passage between the mouth and pharynx is more or less accurately closed by adenoid tissue; the palatal tonsils fit into the glosso-epiglottidean hollows, resting upon the lingual tonsil which floors the passage, and are separated from one another only by the uvula. Now, from all these tonsillar structures leucocytes are constantly passing out into the isthmus faucium, so that it would be difficult for the microbes which normally abound in the mouth (*cf.* Miller, 23) to pass back into the pharynx without coming into contact with them. Numbers of these bacteria of the mouth are, of course, harmless but Ruffer (26) points out that pathogenic organisms are by no means uncommon in the mouths even of healthy subjects, and

while these are not likely in normal circumstances to be absorbed by the buccal mucous membrane, they may cause grave mischief if they pass the tonsils. Metschnikoff and Ruffer have both shown that the leucocytes in the mucus covering the tonsils contain many micro-organisms, and I can fully confirm this. But I think that Ruffer goes too far in saying that "the struggle between micro-organisms and the animal cell is a physiological process always going on *in* the tonsils;" it is probably more accurate to say that this process is always going on *on the surface* of the tonsils. My reason for saying this is that I have stained a large number of sections of tonsils for micro-organisms with Kühne's modification of Gram's method, and with Kühne's universal methylen-blue method, and have had great difficulty in finding a few micro-organisms in the stationary leucocytes lying just under the epithelial lining of the crypts, and that only in one of the tonsils examined, whilst it has been quite easy to demonstrate the Peyer's patches of microbes in the leucocytes from the surface of the tonsil, and in large numbers in the intestine by the same methods. Ruffer himself indeed (*l. c.*, p. 498) tacitly confesses (if I read him aright) that this is so; but I shall allude to this matter again later.

The upper ring of adenoid tissue in the human pharynx surrounds the posterior nares, the entrance to the respiratory tract, and consists of the pharyngeal tonsil, the tubal tonsils (if they are really constant in the human subject), and of the diffuse adenoid infiltration which Bickel and Stöhr (13) describe as running along the floor of the nose and the upper surface of the velum palati. Killian (*l. c.*) has already suggested that the pharyngeal tonsil protects the entrance to the respiratory tract. He points out that the stream of air passing through the nose falls directly on this structure, and he quotes experiments by Wiedersheim, who placed rabbits in an atmosphere full of soot, and found that the soot which had been inspired had lodged mainly in the naso-pharynx. He also presents the interesting suggestion that where animals with wide nostrils (*e. g.*, man and the domestic animals) live much in closed rooms where there are many corpuscular elements in the air, we might expect the pharyngeal tonsil to be large (as is indeed the case in these animals), while in cases where the nose is complicated in form so that the air is purified, or where the stream of air does not fall directly on the pharyngeal tonsil, that organ would not be formed at all, or would at best be small, as it is for instance in the horse. In this connexion I may mention that Ruffer has drawn attention to the fact, that the habits of an animal in regard to food have an influence on the structure of the palatal tonsil. He alludes specially to the number of "macrophages" (or large stationary leucocytes containing microbes) to be found there, but I would point out that the size of the tonsils is possibly influenced by that cause also. The rabbit and guinea-pig, for instance, which live on clean and carefully chosen food, have small tonsils, while

the dog and pig, whose food is of a very mixed description, and which must take in great multitudes of microbes with their food, have tonsils very much larger both relatively and absolutely.

The protection afforded to the alimentary and respiratory tracts by the tonsils is not of course complete, and it must be remembered that these organs are only part of a very extended set of protective appliances. In the alimentary tract, for example, the alkaline saliva acts in the mouth as a germicide for some organisms, and assists the leucocytes furnished by the tonsils. In the stomach the gastric juice is antiseptic. Bunge has indeed gone the length of maintaining that the hydrochloric acid therein contained is almost entirely concerned in keeping bacterial fermentation in check; and the enormous numbers of leucocytes in the adenoid tissue of the stomach and intestine protect the body from the entrance of microbes or their products in those regions. In the respiratory tract the purification of the air in the upper parts of the passages is effected partly by the enclosure of foreign substances in mucus, and the removal of this by the action of the ciliated epithelium, and partly by the action of the leucocytes, which wander out not only through the epithelium of the pharyngeal tonsil, but through the nasal mucous membrane, and through the tracheal and bronchial epithelium from the masses of adenoid tissue which are scattered along the air-passages. In the alveoli, Cox, Tchistovitch, and others have shown that the introduction of any irritant meets with a prompt response in the emigration of leucocytes from the capillaries of the alveolar wall. The tonsils are not, therefore, to be regarded as giving more than a fractional protection,—they are merely parts of an extensive scheme. The faucial tonsils cannot of course deal with the microbes contained in the food which is swallowed,—their action is strictly confined to those which remain in the mouth during the intervals of swallowing, and that action is assisted by the saliva.

The bearing of these physiological facts on the pathology of the tonsils will at once be evident. Stöhr, indeed, realized from the first that the destruction of the tonsillar epithelium might permit the entrance of infective materials into the tissues, and that the very means taken to ward off the attacks of micro-organisms from one part of the body might, under certain circumstances, allow pathogenic microbes or their products more quickly and more surely to enter the circulation; and almost every one who has written on the subject since has alluded to it as a matter of course. I am anxious to show that the matter is not so simple as it at first appears. For example, Ruffer appears to think that the processes of "protection" in the tonsil and in the intestine are identical, whilst it seems to me that there are very considerable differences. I have already said that relatively far more microbes are to be found in a Peyer's patch than in the tonsil, and there are probably two reasons for this. The first is that while the microbes

of the mouth are numerous enough, their number is nothing like so great as that of the organisms in the intestine. The second reason is that the stream of leucocytes from the tonsils is continually passing *outwards* into the mouth, so that it would require bacteria of great strength and persistence to make their way against it into the tonsil in normal circumstances; whilst in the small intestine, though leucocytes do pass through the epithelium in considerable numbers, they do so singly as a rule, and it is comparatively rare to see any such considerable amount of epithelium removed by them there as one sees above the germ-centres in the tonsil; and, moreover, there is a constant *inward* stream of absorption from the intestinal contents, which must surely make it more easy for the microbes to pass through the epithelium than in the tonsil. Generally speaking, one may say that in the upper part of the alimentary tract the protection afforded by the secreted fluids and the emigrated leucocytes is mainly *external* to the body, properly so called, while in the intestine it is mainly *internal*.

Hofmeister (9), Stöhr (25), and others have shown that the mitotic divisions of leucocytes to be observed in adenoid tissue are far more numerous in animals after meals and when they are in good health than when they are debilitated. In the tonsil, therefore, so long as the production of young leucocytes in the germ-centres goes on actively—that is, so long as the animal is in good health—microbes are not likely to gain a footing. If the outward stream so produced is interfered with in any way, however, a local infection from the organisms in the mouth may occur, and according to their nature this may or may not be followed by a general disease. An instance of this, which is familiar to every practitioner, is the frequency with which acute tonsillitis occurs in young girls at or about the menstrual period. Here the cause of the infection is probably the action upon the leucocytes through the blood of the general depression of health, and the same is probably the cause in the cases instanced by Ruffer (*l. c.*, p. 501) of the hospital sore throat which attacks overworked individuals, and of the general diseases which he cites as following tonsillar infection. But it is possible that the production of leucocytes may be as active as ever, and yet that the outward stream may be arrested. Stöhr (7) has shown that in pyopneumo-thorax and allied conditions of suppuration, where there is a large drain of leucocytes from the body, the emigration of leucocytes through the epithelium of the tonsils was brought entirely to a standstill, and the spaces previously formed in the epithelium remained empty. Here, if one may so phrase it, the demand for leucocytes by the lymphatic vessels on the distal side of the germ-centres was greater than usual, and instead of being forwarded through the epithelium, the young cells had to be passed to the other side of the germ-centre to meet the demand. Of course, this would give an excellent opportunity to the buccal

microbes to enter the empty spaces, to pass through the intermediate tissue, and to take advantage of the inward stream to give rise to a general infection by the lymphatics.

There are numerous other points of interest to which I cannot now do more than allude. For example, why is it that the tonsils of young people are relatively so much larger than those of adults? May it not be that their greater liability to infective processes may have to do with it? May not the hypertrophy of the tonsils, so common in weakly children, have reference to their still greater need of protection? And I would suggest, as a question for consideration, whether the deformities of the chest and so on, which are supposed to be due to an obstruction to respiration caused by the enlarged tonsils, may not possibly be caused rather by the constitutional delicacy than by the alteration in the shape of the pharynx.

Conclusions.

1. The tonsils—faucial, lingual, and pharyngeal—are organs arranged to further the reproduction of leucocytes.

2. This reproduction takes place, mainly in the germ-centres, by mitotic division of pre-existing leucocytes.

3. The young leucocytes so formed are partly carried off to the general circulation by lymphatic vessels originating in the tonsil, partly remain in the tonsil as "stationary" cells, and partly wander out into the crypts by perforating the epithelium.

4. They thence pass to the surface of the tonsils, and take up foreign bodies, especially micro-organisms, which would otherwise pass the tonsils.

5. In the human subject the lingual and faucial tonsils, and the slight diffuse leucocyte infiltration of the under surface of the velum palati, form a protective ring or zone between the mouth, which abounds in microbes, and the rest of the *alimentary* tract; while the pharyngeal and tubal tonsils, and the diffuse leucocyte infiltration of the upper surface of the palate, form a protective ring round the upper part of the *respiratory* tract.

6. There is no reason to regard the tonsils as having any absorbent function in normal circumstances; the reproduction of leucocytes is sufficiently active, as a rule, to keep up a continuous outward stream of these cells, and to prevent the entry of foreign substances into the tonsils.

7. Under certain circumstances—for instance, in general debility—the reproduction of leucocytes may be interfered with, and the outward stream of these cells from the tonsils may be arrested. This arrest or other circumstances interfering with the activity of the leucocytes may allow pathogenic organisms from the mouth, etc., to enter the tonsil by the spaces in the epithelium, and these microbes may give rise to a local or general infective process.

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Dr M'Bride thought the paper did not lend itself to discussion, because it was hardly fair to attack the numerous and valuable facts adduced by Dr Gulland by theoretical objections. Dr M'Bride was not a great believer in the palatal tonsils *per se* as a cause of deformities of the jaws and chest; yet when both hypertrophy of the palatal and pharyngeal tonsils existed, such deformities may occur; and undoubtedly, in spite of Dr Gulland's observations, removal of hypertrophied tonsillar tissue is often followed by immediate improvement in the general health. The tubal tonsil is generally imbedded in the cartilaginous portion of the Eustachian tube, and is disseminated. It may be that this distribution of lymphoid tissue assists the ciliary action, preventing the ingress of fœtor-causing organisms into the tympanum. In conclusion, he begged to thank Dr Gulland for his most valuable and instructive paper.

Dr Berry Hayeraft congratulated Dr Gulland upon the care with which he had carried out a difficult investigation, and upon the microscopical preparations by which his paper had been illustrated, which proved that he (Dr Gulland) had made himself

a master in a difficult branch of histological technique. In respect to the protective action of the tonsils there might be some difficulty in accepting part of Dr Gulland's conclusions, for we must not forget the rapidity with which food passes through the pharynx, the fact that living cells (unlike a solution of organic matter) cannot come in at all intimate and rapid relations with food, and also that almost immediately the ingested material is acted upon by the acid gastric juice, undoubtedly an enemy to most organic ferments, and inimical to the leucocytes themselves. In this case the protective action of the tonsils could be only a very local one, and there would be difficulties in the way of regarding these structures as set to guard the alimentary canal and body generally from infective assault. Dr Haycraft also raised a question concerning the development of the tonsils, and the rapidity with which the leucocytes are formed.

Dr James Ritchie said that increase in the size of an organ did not mean an increase of healthy function, often the reverse. He asked Dr Gulland whether the leucocytes in enlarged tonsils exhibited any perceptible evidence of diminished vitality?

Dr Gulland thanked the Society for the attention with which they had listened to his paper, and said, in answer to Dr M'Bride, that he did not mean that hypertrophy of the tonsils had no effect in producing deformity of the chest, but only that more attention should perhaps be paid to the constitutional factor than was sometimes done, that rheumatism and other causes did, of course, allow tonsillitis to occur, but possibly rather by their general debilitating effect than by any local action, and that the tonsil of the Eustachian tubes seemed to be the most variable of all the tonsils in its occurrence, its size, and its position. In answer to Professor Haycraft, Dr Gulland said that whilst the bolus of food in its passage might perhaps press some leucocytes out of the crypts, they could not possibly be mixed fully with the food, and that the micro-organisms swallowed in that way must be dealt with by the gastric juice and the adenoid tissue lower down in the alimentary tract; that the process of breaking down of the epithelium was a slow one in all probability. In answer to Dr Ritchie, he said that the leucocytes produced in hypertrophied tonsils were the same in structure as those produced by the normal tonsils.

2. *Dr Smart* made a communication on CLINICAL EXPERIMENTAL INQUIRIES TO DETERMINE THE INFLUENCE OF DIET AND DRUGS IN THE ELIMINATION OF SUGAR IN DIABETES MELLITUS.

Professor Rutherford congratulated Dr Smart on the value and interest of his communication. His elaborate observations on the influence of different kinds of bread and other articles of diet on the production of sugar in diabetic persons would no doubt be found very valuable in the treatment of the affection. To him as a physiologist, it was of special interest to have had so clearly

shown that sugar and starch are virtually poisons to diabetics. When Dr Smart stated that for every grain of sugar given in the food two or even three grains of sugar were excreted in the urine, and for every grain of starch about a grain and a half of sugar appeared in the urine of diabetics, he indicated that the arrival of sugar in the hepatic cell of a diabetic stimulates in a remarkable degree the pathological process in the cell whereby proteids are wasted by being broken up into sugar and urea, and so thrown away. He had clearly shown the necessity for avoiding whatever could stimulate that morbid process. At the same time, he wished Dr Smart would make observations on the influence of iridin, euonymin, benzoate and salicylate of soda, and other stimulants of the biliary function of the liver in diabetics. Of course they knew that the biliary and glucogenic functions of the liver were very different; still, it would be of scientific interest to know whether or not the pathological glucogenic function of the liver is increased or not by agents that stimulate the biliary function of the hepatic cell.

Dr Smart, replying to Professor Rutherford's observations, said that the urea as well as the sugar had been carefully estimated, and that the results would appear in a future communication. He thanked Professor Rutherford for suggesting a trial of liver stimulants. He would certainly give the remedies named a careful trial.

3. CYSTOSCOPY: NOTES FROM AN EXPERIENCE OF UPWARDS OF FIFTY CASES.

By David Wallace, M.B., F.R.C.S. Ed., M.R.C.S. Eng.

Mr President and Gentlemen,—In December 1889 I had the honour to read at this Society a paper on the cystoscope.[1] I then described the history and mechanism of the instrument, and illustrated its use by reference to several cases of genito-urinary disease which I had examined cystoscopically. To-night I desire to make some remarks from an experience of upwards of fifty cases,—fifty-four in all,—and to describe in detail two or three which seem to me to be both interesting and instructive.

If I leave out of consideration certain cases in which, from one cause or another, the examination was unsatisfactory, owing to the presence of enlarged prostate preventing depression and other movements of the instrument within the bladder; and if I also omit those cases in which nothing more could be discovered by cystoscopic examination than by the other means adopted to account for the patient's symptoms, I find that I have to deal with forty-five cases in which cystoscopic examination was of distinct

[1] *Transactions of the Edinburgh Medico-Chirurgical Society*, vol. ix. 1889-90.

value. These forty-five cases included fourteen cases of tumour, five of calculi, five of tubercular ulceration, eight of cystitis, and five of kidney disease. The other eight cases cannot be classified under the above headings.[1]

The tumours, fourteen in number, varied in their character. In twelve operative interference was had recourse to, and of these five were papillomata, one a leio-myoma, and eight villous cancers. In the other two no operative interference was attempted. Four of these cases very vividly exemplify the diagnostic power of the cystoscope in obscure genito-urinary disease, and I therefore propose to give an account of them.

Case I.—J. A., male, æt. 60, under the care of Mr Duncan, complained of *hæmaturia* of some weeks' duration. The urine was very dark and contained much blood, some of which was passed in the form of large irregular clots. The clots were passed during the flow of the urine. No symptoms of the presence of stone or kidney mischief. On sounding nothing was felt. Microscopically epithelial squames were seen, but no cancer cells recognisable as such.

Diagnosis uncertain, but hæmaturia, probably vesical or prostatic in origin.

Cystoscopic Examination.—The bladder washed out, and six ounces of boric lotion injected. The bladder wall not rugose. On left infero-posterior part of bladder above the left ureter an irregular villous tumour seen. The tumour had a narrow base of attachment, and projected into the bladder for about an inch and a half. There was some extravasated blood on its surface. In this case a second examination was made, when the appearance above noted was again satisfactorily observed.

Diagnosis.—A pedunculated villous tumour about the size of the terminal phalanx of thumb.

In this case suprapubic cystotomy was performed by Mr Duncan. He found the tumour in position and character similar to the idea derived cystoscopically, and was able to remove it, with the best possible result.

Case II.—Wm. D., male, æt. 30, under Prof. Chiene's care. Gave history of *intermittent hæmaturia* of several months' duration. No evidence of an abnormal vesical condition could be gained on thorough investigation by sounding, although there was apparently some roughness on the floor of the bladder. Microscopical examination of the urine gave no evidence of tumour growth.

Cystoscopic Examination.—Eight ounces of fluid injected. On the floor of the bladder an irregular blood-clot of some size was seen. Laterally the bladder quite healthy in appearance; but a round, smooth, non-pedunculated swelling the size of a walnut was discovered in the middle line of the wall anteriorly.

[1] In these eight cases there were three of enlarged middle lobe of the prostate, and two of ulceration of the bladder wall.

Diagnosis.—A smooth sessile tumour situated on anterior aspect of bladder wall.

Prof. Chiene performed suprapubic cystotomy. The incision to open into the bladder in the middle line, passed into the tumour, so that another incision had to be made a little to one side. When the bladder was opened into, a portion of the tumour was found under the mucous membrane, but not projecting into the cavity, while the portion seen cystoscopically was found to be as described on cystoscopic examination. The tumour was easily shelled out and removed. On microscopical examination it proved to be a leio-myoma, one of the rarer forms of bladder tumour.[1] The patient made an excellent recovery.

It is interesting in this case to note that even after the presence and exact position of the tumour was seen, it could not be felt with the sound or bimanually, *i.e.*, with one finger in the rectum and the other hand above the pubis.

Case III.—Mrs G., æt. 64, under the care of Dr Barbour. Gave history of *intermittent hæmaturia*, *frequency of micturition*, and *dysuria*. The two last symptoms were of one year's duration. On bimanual examination a soft swelling could be felt between anterior fornix and abdominal hand, situated a little to the left of the middle line. Microscopic examination of the urine gave no evidence of a neoplasm.

Cystoscopic Examination.—Eight ounces of lotion injected. The bladder as a whole healthy, but on the right inferior aspect, just at position of the right ureteral opening, a villous tumour seen. Base of attachment as broad as tumour surface, but the tumour projected into the bladder for about one inch. The tumour obscured the right ureteral opening, but the left could be seen as a narrow fissure.

Diagnosis.—A sessile villous tumour about the size of the terminal phalanx of the thumb.

Dr Barbour afterwards dilated the patient's urethra, and made a digital examination verifying the position, size, and attachment of the neoplasm. He found, however, that it infiltrated the bladder wall to such an extent that operative interference seemed inadvisable. A small portion of the tumour which he removed was found microscopically to be carcinomatous.

Case IV.—J. H., male, æt. 53, under the care of Dr Robert Stewart. Gave history of *intermittent hæmaturia* of three or four years' duration. The condition was not benefited by treatment, and occasionally the bleeding was alarmingly profuse. The bladder had not been sounded, but masses of membrane passed per urethram were found to consist in part of epithelial cells.

Cystoscopic Examination.—Eight ounces of fluid injected. A perfect view of the whole bladder was obtained, when it was seen anteriorly and laterally to be smooth and healthy, although paler

[1] Billroth, *Surgical Pathology.*

than usual. (He had a week or two previously had very profuse and alarming bleeding.) From the floor of the bladder there projected a well-marked and extensive villous tumour. The tumour consisted of numerous long wavy fimbriæ, which were very vascular. The bloodvessels in the villi were distinctly visible. The tumour was sessile, attached to the floor of the bladder, close to its neck, for about half of its circumference, but the mass of it was a little to the right.

Diagnosis.—A sessile villous tumour.

In this case supra-pubic cystotomy was performed by Prof. Chiene and the tumour removed. Its attachment and character were as described cystoscopically. It was found on microscopical examination to be carcinomatous, but probably an example of a tumour originally benign which had become malignant.

The above four cases of vesical neoplasm are very typical examples of what I have found in the thirteen cases which I have had the opportunity to observe. Cases I. and II. show very markedly that, even by the most careful local examination in the hands of the most experienced surgeons, no evidence whatever can be obtained of the presence of a neoplasm in some cases of hæmaturia. Accordingly, to diagnose with certainty the presence of a tumour growth it is necessary either to examine the bladder cystoscopically or to have recourse to digital exploration; and when considering the latter, the surgeon must face the difficulty that frequently hæmaturia is due to tubercular ulceration of the bladder or disease of the kidney, so that unless these be excluded by other evidence, the operation of cystotomy may have been deemed necessary for diagnostic purposes when cystoscopic examination would have sufficed.

These cases further show, I think, that accurate diagnosis may be made by the cystoscope—accuracy which cannot be otherwise arrived at by any other examination short of digital exploration after cystotomy. Again, if the examination with the cystoscope fails to be satisfactory, and the condition causing the symptoms be not determined, cystotomy can always be resorted to.

Calculi.—It is admitted that the detection of a calculus by the sound may be rendered difficult in various ways, as, for example, by the lodgment of the calculus in a sulcus or sac of the bladder, or when the stone is small in a depression between rugæ, or by a fold of mucous membrane partly covering it. That such difficulties exist is shown by the various modifications of the sound introduced with the object of aiding the detection of calculi. Only a month or two ago a new sound, introduced by Sir Henry Thompson, to enable a small calculus to be more easily detected, was figured in the *British Medical Journal*. Cases have been recently quoted by two surgeons of encysted calculi missed by the sound, but detected

by the cystoscope. I have not been fortunate enough to have such a case, but I have had two cases in which, after very careful examination, the surgeon having failed to detect the presence of stone, I was able to demonstrate its presence by the cystoscope.

Case V.—J. B., male, æt. 65. Patient stated that he had passed many small stones. History of present illness as follows:—Had suffered for last few weeks from severe pain in the loin. A few days before examination the pain came on suddenly and passed away suddenly, but was very severe while it lasted. He had not recently passed any calculi by the urethra, but complained of the various symptoms which point to the presence of a stone in the bladder. After very careful examination by sounding, no stone was detected. A lithotrite was not used.

Cystoscopic Examination.—Six ounces of fluid injected. The bladder wall was healthy in appearance, and not rugose. An elongated, almond-like body, dark in colour and tuberculated, was seen on the floor of the bladder. It moved when the patient shifted his position, and on directing the beak of the cystoscope so as to touch it, it was readily proved to be a calculus.

Diagnosis.—A stone about the size of a horse-bean, probably uric acid in composition. The stone was afterwards removed.

The fact is, patients who suffer from calculi come before the surgeon nowadays at a very early period after symptoms of the presence of a stone have been manifested,—so soon, indeed, that not many months ago a discussion was raised by two eminent surgeons when a concretion should be dignified by the term calculus or stone, the one fixing the limit at three or four grains, the other at twenty. In rugose bladders there is the risk of such small calculi getting into rugous depressions and being missed on sounding. With the cystoscope, when carefully used, the detection of such small stones is more probable; and accordingly, in cases of suspected stone, where sounding proves negative, I think cystoscopy should be had recourse to.

Tubercular Ulceration.—A patient who suffers from hæmaturia accompanied by pyuria may be the subject of different forms of genito-urinary disease which have to be differentiated from each other; and although the history, presence of tubercular bacilli, and the symptoms from which the patient suffers, may cause the diagnosis to be almost certainly tubercular, it is nevertheless very difficult in some cases to localize the disease. Is the condition entirely prostatic, or is it vesical, or renal, or a combination of any two or all three? In a paper[1] read before this Society by Dr R. W. Philip, the author points out that there may be a comparative absence of symptoms at the outset of tubercular affection of the

[1] R. W. Philip, "Case of Tubercular Ulceration of Bladder," *Trans. Edin. Med. Chir.*, vol. viii., 1888-89.

genito-urinary system, and further, that even in the presence of a grave bladder lesion there may be persistent absence of cystitis, or even discomfort in micturition. He also refers to the difficulty of detecting tubercle bacilli in the membranes passed per urethram. The two following cases exemplify what important help may be derived from cystoscopic examination where the case is doubtful.

Case VI.—Miss F., æt. 24. Recommended to me by Dr James Ritchie. The patient, a robust, healthy-looking woman, complained of hæmaturia and pain in the loins and bladder. Pain was first felt in the bladder four years earlier, but at that time there was no bleeding. She observed blood in the urine for the first time two months before I examined her. She had pain during and after micturition. There was great frequency, both during the night and day. Microscopically blood and pus in the urine, no crystals nor cells. Tubercle bacilli looked for, but not observed. There was a very decided family history of tubercular disease.

Cystoscopic Examination.—Six ounces of fluid injected. The greater part of the bladder wall healthy in appearance, but a regular, shallow, dark-coloured area, rather larger than a sixpence, seen just above the right ureter.

Diagnosis.—An ulcer, probably, from history, tubercular.

Under local and general treatment the patient got completely better.

Case VII.—T. B., male, æt. 37, under care of Prof. Greenfield. Patient had a perinephritic abscess on left side, which burst into the pleura, from thence opened into a bronchus, and was thus discharged. Prostate completely destroyed by tubercular disease. When examined the patient had pain over the left kidney and suffered from frequency of micturition. Urine contained pus and blood—pus in large amount.

Cystoscopic examination made, after injection of six ounces of fluid, with much difficulty, but pus was observed with great distinctness to be passed from the left ureteral opening. Nothing further could be made out, owing to restlessness of the patient.

In Case VI. it was difficult to say, before cystoscopic examination, whether the condition was vesical or renal, but I think the examination showed definitely that the condition was vesical, and the after history may be said to have proved it to be so. Case VII. is of great interest, as it shows that you can see pus passing from the ureteral opening, and when taken together with the next case (Case VIII.) indicates how important cystoscopic examination may be in doubtful cases of pyuria or hæmaturia.

Case VIII.—A. B., male, æt. 37, under care of Prof. Fraser. Gave history of *intermittent hæmaturia*. Microscopically no

tumour cells found in the urine. The patient attributed his condition to an injury to the loins sustained by him some months previously.

Cystoscopic Examination.—Eight ounces of fluid injected into the bladder, which was not rugose nor unduly injected. No abnormality of the bladder observed, but while clear urine was seen to be ejected from the right ureteral opening, a brownish red fluid issued from the left and gradually diffused through the contents of the bladder.

Diagnosis.—Blood from the left kidney the cause of the blood in the urine. The bladder is healthy, and from cystoscopic examination there is no reason to suspect any disease of the right kidney.

It is a fact that by cystoscopic examination urine, blood, or pus can be distinguished passing from the ureteral opening or openings, and in cases where you find the bladder healthy, and see blood or pus ejected from one or both openings, you have distinct evidence that there is disease of the kidney or kidneys. In obscure cases, therefore, we may be able to decide that one or both kidneys is diseased, and, acting on the knowledge thus gained, can attack the diseased organ. Such knowledge is of much value in considering the operation of nephrectomy. It is on record that a nephrectomy has been performed when the kidney removed was the only one present; but, apart from such a rare occurrence as that, we must recognise that a favourable result after nephrectomy greatly depends on the condition of the kidney which is left. Is it in a condition to take on the extra work thrown upon it by the removal of the other? or is it so diseased that it is unable to perform more than its own share of the work? This point may have some light thrown upon it by cystoscopy; for by cystoscopic examination you would at least be able to judge, by observing what passed from the ureters, whether the kidney intended to remain was likely to be functionally active.

Now let me pass, first, to a consideration of the points to be attended to for satisfactory cystoscopic examination; and, second, to a short *résumé* of the value of the instrument. The points to attend to in the use of the instrument are very simple, but I may very shortly refer to some of these, which are of most importance to enable us to gain a satisfactory result.[1]

1. Precautions should be taken to avoid the risk of a chill to the patient, just as in any other examination of the bladder.

2. The urethra must have a calibre sufficient not merely to allow the introduction of the instrument, but also to admit of free movement of the instrument within the bladder.

3. It is imperative that the bladder should contain at least six ounces of clear fluid, and that the patient, unless he be able to bear

[1] In my previous paper (*op. cit.*) I pointed out how a cystoscopic examination should be made, but I wish to draw particular attention to the points now taken up.

the introduction of the fluid and movement of the instrument without pain, be either locally or generally anæsthetised. I have examined most of my cases without any anæsthetic, and in the majority I have found that the examination caused little or no pain. I think the chief indication for an anæsthetic, local or general, is to be derived from the quantity of urine which the patient can comfortably retain. If he is able to retain eight ounces without discomfort, no anæsthetic is, as a rule, required; but, on the other hand, if the patient be very nervous, it is better even in such circumstances to administer an anæsthetic.

4. The examination must be methodically and carefully made, so that the whole of the interior of the bladder may be thoroughly investigated. In connexion with the time required for the examination, it is interesting to note that the superficial area to be examined in a bladder which contains eight ounces of fluid is as follows—When the bladder is ovoid, 30 sq. inches; when the bladder is spherical, 27·27 sq. inches. The field of the cystoscope is about half a square inch. I prefer to use the "Anterior" instrument, as its sphere of usefulness is much greater than that of the posterior. The whole bladder wall may be examined by the "Anterior," except a small part opposite the opening of the urethra into the bladder; for the examination of it the posterior instrument is required.

5. Most affections of the bladder are situated in the inferior third, and it is to that part, therefore, that particular attention should be directed. In every case the urethral openings should be carefully observed. We must not forget, however, that not only may there be ulceration of the roof of the bladder, as in Dr Philip's case, already referred to in this paper[1]; but, as in Case II., which I have related, tumour growths may spring from the anterior aspect. The ureters, unless obscured by tumour growth, can, as a rule, be easily brought into view; and when the contained fluid of the bladder is clear, it is not difficult to see the urine being ejected from their orifices.[2] Cases VII. and VIII. exemplify very beautifully how pathological conditions may in this way be observed.

6. When an abnormality has been discovered, the observer requires to move the beak of the instrument into different positions to determine exactly what he sees. I can illustrate the value of this by reference to two errors in diagnosis which I have made. The first was a case in which I readily found a calculus, but was deceived regarding its size,—I thought it larger than it turned out to be. To have gauged the size correctly all that was necessary

[1] V. *op. cit.*

[2] In a case of extroversion of the bladder which I recently had an opportunity of observing, the urine was ejected from the ureters for a distance of 4 to 6 inches when the patient was excited. Ordinarily, however, it merely trickles at irregular intervals from the urethral openings.

was to approach or recede from the stone until its surface was most distinctly seen. At the time when I got the clearest definition, I would have determined the actual size. In the second case, the patient had repeatedly passed gravel, and gave a very distinct history of stone symptoms. Professor Chiene failed to detect any stone or other abnormality by careful sounding. I examined the patient cystoscopically, and on the floor of the bladder a little to the right of the middle line readily distinguished an irregular body about the size of a Barcelona nut. I described the condition as follows:—To the right of the middle line on the floor of the bladder there is a grayish-white tuberculated body about the size of a Barcelona nut. *Diagnosis*—A calculus, probably formed of oxalate of lime coated with phosphates. The examination pained the patient considerably, and I was so satisfied of the diagnosis that I merely held the instrument in the same position to allow two or three medical men who were present to see the calculus. A few days afterwards the patient was put under chloroform, the urethra was dilated, and an endeavour made to remove the calculus. For its removal small lithotomy forceps were introduced, but after grasping the abnormal object and attempting to withdraw it, it slipped from between the blades, and only portions of phosphatic deposit came away. This happened twice, and an error of diagnosis was suspected; the urethra was further dilated, and the finger introduced, when the supposed calculus was found to be a small fibrous tumour coated with phosphates. It was easily removed by scraping with the finger nail. In this case more careful examination would have shown that the body seen was attached immovably to the bladder wall, the beak of the instrument could have been turned so as to touch it, and the error in diagnosis have been avoided. On reference to Hurry Fenwick's book, I note that he warns us against making such mistakes, and quotes a case very similar to the one I have described.[1]

I think the numerous recorded cases where affections of the bladder *per se* had not been recognised, or where pathological conditions in the urine had not been correctly interpreted until a cystoscopic examination had been made, clearly prove that frequently all investigation, short of cystotomy, without cystoscopy, may be inadequate to discover the source of the patient's ailment.[2] Indeed, I may safely say that nothing short of *feeling directly* with the finger, or *seeing* the abnormal condition, can enable the surgeon to give a positive or negative diagnosis regarding the presence or absence of some abnormal conditions in the bladder, *e.g.*, tumour or ulcer.

Just as after the removal of a tumour from the body it is frequently impossible to say what its character is until it has been

[1] *The Electric Illumination of the Bladder and Urethra.* By Hurry Fenwick, F.R.C.S. Second edition. 1889.

[2] Papers by Nitze, Hurry Fenwick, Berkeley Hill, Bruce Clarke, etc.

microscopically examined, so with cystoscopic examination,—by it we may observe a pathological condition of the bladder, but we are not yet able to interpret correctly whether in the case of a tumour it is malignant or benign. As we gain more experience, however, of the appearances observed when a tumour is *in situ*, and later have its pathology microscopically determined, we will be better able to give a prognosis as well as a diagnosis.

In the paper which I read before this Society in December 1889, I said, "I think we are justified in the belief that the cystoscope *will prove* a most valuable aid in diagnosis," and I then quoted Sir Henry Thompson, who wrote in the *British Medical Journal* of April 1888 in the following terms:—"Although we should not give up in any respect the simple means of prosecuting diagnostic research hitherto employed, by all means keep the cystoscope in reserve for certain exceptional cases, where other usual methods have been tried and have proved unsuccessful." To-night I am able to say, after a much greater experience of the instrument, that the cystoscope *is of the greatest* value in the diagnosis of genito-urinary disease, and that the surgeon in any case in which he is doubtful of the diagnosis, fails to use all the means at his disposal to form a diagnosis, if he does not attempt to discover the pathological condition by means of its use before he operates. With the cystoscope we are able not merely to say that there is or is not disease of the bladder; but in cases where the condition is due to renal disease, we can demonstrate that the disease is renal, and also which kidney is affected.

I do not think it necessary to examine the bladder cystoscopically in all genito-urinary disease; but careful consideration of the numerous cases which I have now had an opportunity of examining makes me emphatically of opinion that when other means have failed to enable the surgeon accurately to determine the condition from which the patient suffers, cystoscopic examination should be had recourse to before treatment is entered upon or operative interference recommended.

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